“ONE STEP APPROACH FOR DIABETES MELLITUS IN PREGNANCY”

DISSERTATION SUBMITTED IN FULFILMENT OF THE

REGULATIONS FOR THE AWARD OF

MS OBSTETRICS AND GYNAECOLOGY

DIVISION OF OBSTETRICS AND GYNAECOLOGY

PSG INSTITUTE OF MEDICAL SCIENCES AND RESEARCH

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

GUINDY,CHENNAI,TAMILNADU,INDIA

Reg. No : 221216453

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Certificate

CERTIFICATE

This is to certify that **Dr. SHRUTHII NANJUNDAPPAN** Reg. No: 221216453 has prepared this dissertation entitled **"ONE STEP APPROACH FOR DIABETES MELLITUS IN PREGNANCY".** under my overall supervision and guidance in the Institute of PSG Institute of Medical Science and Research, Coimbatore in partial fulfilment of the regulations of Tamil Nadu **Dr. M.G.R Medical University** for the award of **M.S. Degree in Obstetrics and Gynaecology.**

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Declaration

DECLARATION

I hereby declare that dissertation entitled **"ONE STEP APPROACH FOR DIABETES MELLITUS IN PREGNANCY"** was prepared by me under the guidance and supervision of **Dr. SEETHA PANICKER MD DGO., DNB,** PSG Hospitals Coimbatore.

The dissertation is submitted to the Dr. M.G.R. Medical University in partial fulfilment of the University regulations for the award of MS degree in Obstetrics and Gynaecology. This dissertation has not been submitted for the award of any Degree or Diploma.

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DR. SHRUTHII NANJUNDAPPAN

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Introduction

INTRODUCTION

Diabetes mellitus is a disorder of carbohydrate metabolism characterized by high glucose levels ,as a result of defect in insulin production, or its action nor both. The prevalence of diabetes worldwide has increased significantly in the last three decades, reaching almost epidemic proportions in south Asia. According to World Health Organisation estimates, India has the highest number of cases of Diabetes in the world. As estimated 31.7 million people with diabetes in 2000 in India are projected to increase to 79.4 million in 2030 (1).The prevalence reported from various parts of the country ranges from 2-4% in rural to 10-16% in urban population. A survey done in urban India in 1986 did not find any case of diabetes in less than 30 yrs of age(2) ,but 15 yrs later, National urban Diabetes survey (2001)reported a prevalence of 5.4% in under 30 age group(3).WHO prevalence in India 16.55%(4)

Before the discovery of insulin, with uncontrolled diabetes infertility was the rule .Many of these women were amenorrhoeic and only about 2% of diabetic patients conceived. The ones who conceived had high risk of morbidity and mortality. The immediate maternal

mortality was around 25%, a few dying in pregnancy but with majority in puerperium, giving an overall mortality attributed to pregnancy in diabetes was 50% and perinatal mortality was high as 60%.

The glycemic control during pregnancy is due to the compensatory hyperinsulunemia as there is insulin resistance during pregnancy,as the antenatal women cannot increase the insulin secretion so that she may overcome the insulin resistance is when she develops GDM. In short when the pregnant mothers beta cell function cannot overcome the antagonism created by the anti insulin hormones normally secreted during pregnancy.

Approximately 50% of women with gestational diabetes will develop type II diabetes later in life.

Our study is to compare the Diabetes in Pregnancy Study Group India(DIPSI) recommendation which is a single 75gm oral glucose blood test taken 2 hrs later irrespective of last meal with the World Health Organisation (WHO) recommendation which is a two step process of FBS followed by 75gm oral glucose blood test 2 hr later.

Review of Literature

REVIEW OF LITERATURE

1. HISTORIC PERSCEPTIVE :

Diabetes Mellitus is one of the oldest diseases of mankind. Inspite of the all the available data , Gestational Diabetes Mellitus was poorly mentioned and studied at least till nineteenth century.

The credit for naming Diabetes goes to a Greek physician, Aretaeus of Cappadocia, (30-90 AD) . The Hindu medical writings of the 6th century refer to diabetes as honey urine. Mathew Dobson (1776) of Liverpool gave first description of hyperglycemia. John Rollo from Edinburgh (1809) was the first to apply the adjective 'mellitus' derived from the Greek and Latin word meaning honey. Claude Bernard (1813-78) clarified glucose metabolism.

First case report on diabetes in pregnancy was that of Bennewitz in 1826. In 1882 Mathew Duncan presented a paper entitled 'Puerperal Diabetes'. Benedict (1884-1936) discovered the tests for glucose in Urine and blood.

Fedric Banting and Charles Best of University of Toronto discovered Insulin in 1921, which changed the outlook of diabetic

pregnant women. The term insulin was coined by Macleod in 1922. Belgian researcher J P Hoet published a study on 'carbohydrate metabolism during pregnancy' and first used the term ' Metagestational diabetes' in1954.

O Sullivan and Mahan worked on glucose screening test and derived their figures from a major project on maternal and fetal medicine in 1950's initiated by the Boston city hospital . Jorgen Pederson was the first to use the modern term 'Gestational Diabetes' in 1967 which was later on promoted by Dr. Norbert Freinkel.

HbAIc increase in diabetes was first described in 1969 by Samuel Rahbar.

In 1973 O 'Sullivan et al proposed the use of 1 hour screening test whole blood glucose of 130 mg/dl was considered a positive screening test.

Carpenter and Coustan established the screening test for GDM, concluded that 1hour post glucose plasma test is superior over other tests for routine GDM screening. They renewed the NDDG, three hours OGTT criteria and modified it with lower threshold points. These lower

points were derived from the use of more specific enzymatic assays in blood sugar determination.

Haworth JC in 1975 studied effects of abnormal glucose tolerance in pregnancy on infant Mortality rate and Morbidity and found that the glucose intolerance in the mother is at risk for hypoglycemia in the fetus. The use of HbAIc for monitoring the degree of control of glucose metabolism in diabetes patients was proposed in 1976 by Koenig of coworker.

GDM as a clinical entity officially began in 1979 when the NDDG issued an update classification of diabetes types. In 1979, the first International workshop conference on GDM also met, essentially declared GDM as a disease, finding it at a significant health risk that needed treatment & instead of the more neutral carbohydrate intolerance of pregnancy, the term ‘Gestational Diabetes Mellitus’ was used.

2. CARBOHYDRATE METABOLISM DURING PREGNANCY

The most important reason why pregnancy exacerbates the diabetic tendency of asymptomatic women is the progressive increase in insulin resistance that occurs during gestation. Other reasons for this diabetogenic tendency are the increased lipolysis and the alterations in gluconeogenesis which normally occur during gestation.

During the first and early part of the midtrimester there is increased sensitivity to insulin and diabetic patients have a tendency towards hypoglycemia. This enhanced insulin sensitivity is probably due to the high levels of estrogen. The opposite occurs in the third trimester when a given dose of insulin has a decreased hypoglycemic effect.

2.1 Diabetogenic effects of pregnancy

Insulin resistance

- Production of human placental lactogen
- Increased production of cortisol, estriol, and progesterone
- Increased insulin destruction by kidney and placenta

Increased lipolysis

- The mother utilizes fat for her caloric needs and saves glucose for fetal needs

Changes in gluconeogenesis

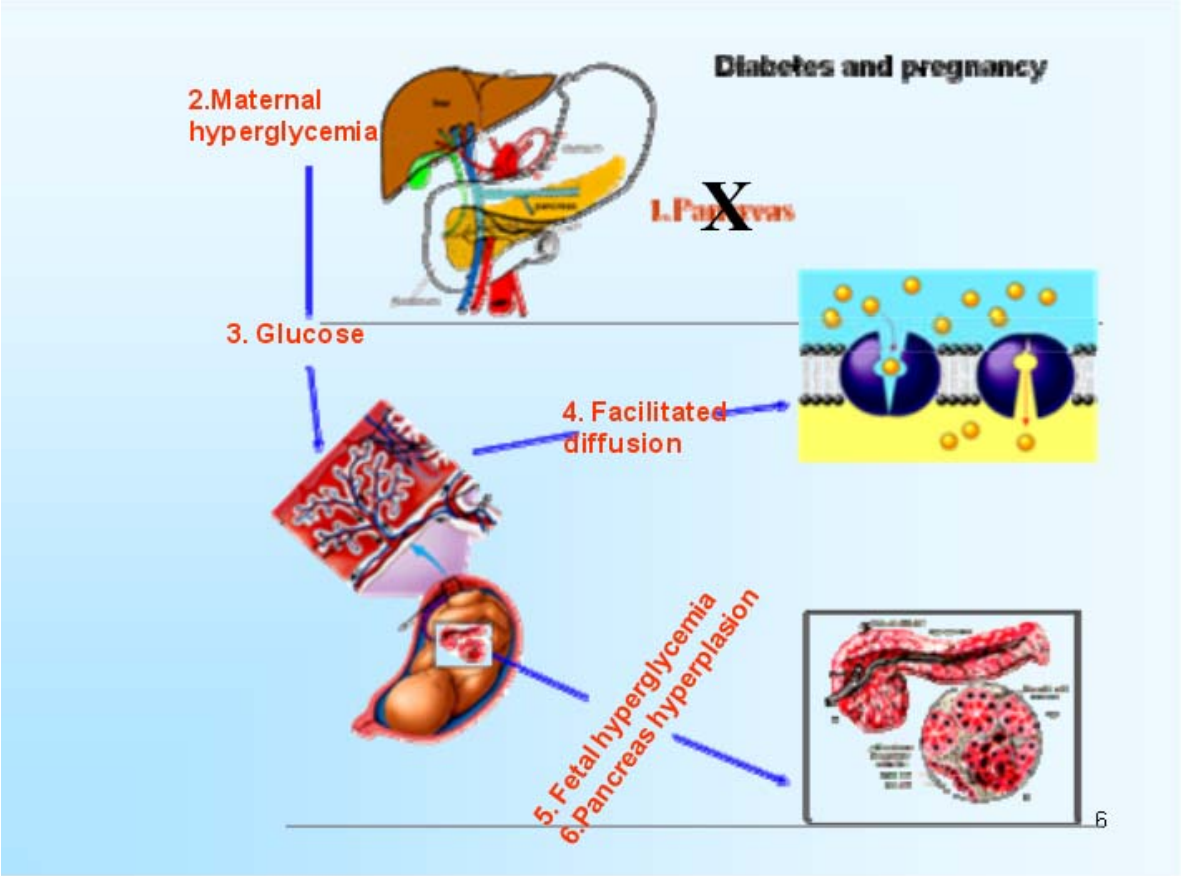
- The fetus preferentially utilizes alanine and other amino acids, depriving the mother of a major neoglucogenic source

This increased insulin resistance stems mainly from the antagonistic effect of human placental lactogen. Accelerated insulin catabolism by renal and placental insulinases and the anti-insulin effects of other hormones (cortisol, estriol, progesterone) produced in large amounts during pregnancy also contribute to insulin resistance. The increased insulin resistance in the third trimester explains why gestational diabetes is more common after 26 weeks.

It also explains the increased risk for ketoacidosis in pregnant women with type I diabetes.

As a result of the physiologic changes of pregnancy, the normal fasting blood sugar is 65 ± 9 mg/dl. The mean nonfasting blood sugar level is 80 ± 10 mg/dl. Postprandial elevations normally never exceed 140 mg/dl (1)

Diabetogenic Effects Of Pregnancy



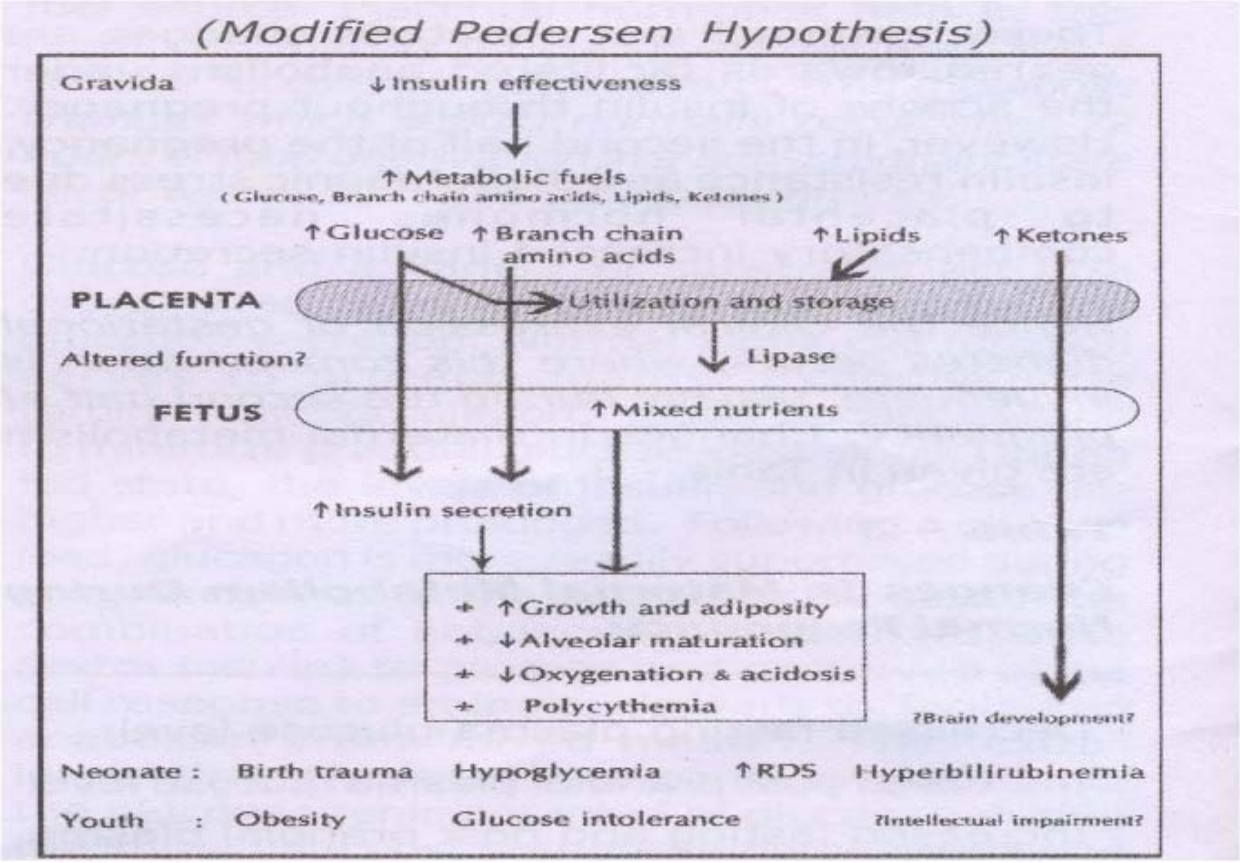
2.2 Fetal Glucose Homeostasis

Glucose crosses the placenta by facilitated transport . The fetal glucose levels parallel maternal levels, but it is about 10 mg/dl lower compared to the mother. Maternal insulin does not cross the placenta and the fetus produces its own insulin from late first trimester. However, the fetal insulin response in a normal pregnancy is sluggish, and it is likely that fetal insulin plays less of a role in glucose homeostasis, and more in promoting growth.

Pedersen proposed the theory of " hyperglycaemic-hyperinsulinism". According to this, maternal hyperglycaemia leads to fetal hyperglycaemia, which in turn stimulates fetal pancreatic β-cells hypertrophy leading to fetal hyperinsulinamia. The fetal hyperinsulinaemia is responsible for the increased fat deposition and macrosomia, organomegaly, especially of the liver and the heart, increased erythropoietin production and decreased surfactant production. The consequences of these effects are an increased risk of birth trauma and intrapartum asphyxia, respiratory distress syndrome and polycythaemia results form the direct effect of high insulin levels in the fetus. In a nutshell, if the maternal glucose levels can be maintained in a normoglycaemic range, the adverse

effects of fetal hyperinsulinaemia can be minimized and the fetal and neonatal outcome improved.

Modified Pedersen's Hypothesis



3. EFFECTS OF DIABETES ON PREGNANCY

The great majority of women with carbohydrate intolerance during pregnancy do not have signs or symptoms. Unfortunately, carbohydrate intolerance during pregnancy causes significant increases in fetal and maternal morbidity.

3.1 Effects of diabetes on the mother (2)

Preeclampsia

- Affects 10-25% of all pregnant diabetics Infection
- High incidence of chorioamnionitis and postpartum endometritis

Postpartum bleeding

- High incidence due to exaggerated uterine distension

Cesarean section

- High incidence of pregnant diabetics

The incidence of preeclampsia is approximately 15% and it is associated with poor glycemic control and end-organ damage (3). Among the fetal effects, the frequency of congenital abnormalities is increased in women with poorly controlled type I diabetes and the incidence of fetal macrosomia is increased in women with

gestational and type II diabetes. Fetal growth restriction is common in women with type I diabetes and end-organ damage.

3.2 Diabetics with End-Organ Damage

3.2 (a) Diabetic nephropathy

The characteristic features of patients with diabetic nephropathy are the presence of proteinuria and hypertension in the first or second trimester of pregnancy. Also, these women are at particularly high risk of developing superimposed preeclampsia, which affects 50% of pregnant diabetics with renal disease. The main fetal disorders occurring in patients with diabetic renal disease are prematurity and fetal growth restriction. The incidence of preterm delivery in these patients is approximately 45%. Fetal growth restriction affects approximately 20%.

(b) Diabetic retinopathy

Diabetic retinopathy affects approximately 40% of all insulin-dependent diabetics. 80% of the cases, “background retinopathy.” 20% of these patients have neovascularization along the retinal surface, and this is named “proliferative retinopathy.” The important group to identify is the latter because the new vessels are fragile and may bleed profusely with the changes in

intraocular pressure that occur during labor, leading to sudden vision loss. Therefore, labor is contraindicated in these patients because Valsalva efforts may increase intraocular pressure, causing vitreal hemorrhage and retinal detachment.

(c) Diabetic neuropathy

Gastroparesis vomit continuously and frequently develop starvation ketosis. Treatment is by intermittent gastric intubation or from the administration of metoclopramide or erythromycin.

(d) Diabetic cardiomyopathy

Prognosis is poor. Management with cardiac-obstetric care unit.

(e) Metabolic Syndrome

The metabolic syndrome is age dependent and in USA it is reported in 7% of individuals between 20 and 29 years of age and in 44% of those aged 60-69 years (4)

Definition of the metabolic syndrome according to the World Health Organization and the National Cholesterol Education Program Adult Treatment Panel III (NCEPATP III)

e (i) World Health Organization

- Diabetes, impaired glucose tolerance, impaired fasting glucose and/or insulin resistance

Plus at least two of the following:

- Abdominal obesity (waist to hip ratio > 0.85 in women or > 0.9 in Men and/or body mass index > 30kg/m2)
- Triglycerides > 150 mg/dl and/or HDL < 40 mg/dl in women or < 35 mg/dl in men
- Blood pressure ≥ 140/90 mmHg
- Microalbuminuria: urinary albumin excretion ≥ 20 ug/minute or albumin to creatinine ratio ≥ 30 mg/g

(ii) NCEPATP III

At least three of the following:

- Fasting plasma glucose > 110 mg/dl
- Abdominal obesity (waist circumference > 35 in. in women or > 40 in. in men)
- Triglycerides > 150 mg/dl; HDL < 50 mg/dl in women or < 40 mg/dl in men Blood pressure ≥ 130/85 mmHg

(f) Diabetic ketoacidosis

DKA is a serious emergency that requires adequate treatment to save maternal and fetal lives. DKA results from a deficit in insulin and the response to that deficit by counter-regulatory hormones. As a result of decreased cellular glucose consumption and increased neoglucogenesis, the blood sugar concentration reaches high levels. This severe hyperglycemia causes osmotic diuresis with depletion of the intravascular volume and electrolyte changes. Simultaneously, the cells start to use fatty acids as a source of energy (lipolysis) with production of ketoacids that consume the body buffers, resulting in a high anion gap and metabolic acidosis. If uncorrected, this may lead to maternal and fetal death. This emergency requires early diagnosis and aggressive treatment with identification and elimination of the precipitating event.

(g) Fetal macrosomia

Fetal macrosomia, defined as an estimated fetal weight (EFW) equal to or larger than 4000 g. Usually the first indication of developing macrosomia is an abdominal circumference larger than other measurements, resulting in abnormally elevated head to abdomen and femur to abdomen ratios. The EFW may be in the

60^{th} - 80^{th} percentile between 26 and 32 weeks but by the end of the pregnancy will be above the 90^{th} percentile.

The positive predictive value for the detection of macrosomia exceeds 90% when the abdominal circumference or the EFW is above the 95^{th} percentile.

(h) Detection of Diabetic Embryopathy

There is good evidence indicating that levels of HbA1C greater than 8.5% are associated with a 20-25% probability of fetal developmental abnormalities.

h(i). Most common congenital abnormalities in infants of diabetic mothers

Central nervous system

- Anencephaly
- Holoprosencephaly
- Encephalocele

Heart and great vessels

- Transposition of the great vessels
- Ventricular septal defect
- Aortic coarctation
- Atrial septal defect

Skeletal and spinal

- Caudal regression syndrome (1.3/1000 diabetic pregnancies)

Genitourinary

- Renal agenesis
- Ureter duplex

Gastrointestinal

- Anal atresia

First trimester screening for aneuploidy, nuchal translucency, plus the measurements of free beta-HCG and PAPA-A. Evaluation of the maternal serum alpha-fetoprotein (MSAFP) at 16 weeks to screen for open neural tube defects. An abnormal MSAFP indicates the need for comprehensive ultrasound examination of the fetal spine and in a few cases the need for genetic amniocentesis. Irrespective of the results of the first and second trimester screening, should have a detailed anatomical survey of the fetus or comprehensive ultrasound examination at 18-20 weeks of gestation. The fetal survey must include a fetal echocardiogram.

(i) Stillbirth

In gestational diabetes associated with fasting hyperglycaemia > 105 mg/dl, There is an increased risk of fetal death in the last 4 to 8 weeks of pregnancy.

The mechanism by which maternal hyperglycaemia operates to cause fetal hypoxia is via fetal hyperglycaemia, which results in stimulation of fetal pancreatic β-cells and fetal hyperinsulinaemia.

(j) Preterm Labour

The risk of spontaneous preterm births was 28% higher in women who were screen positive but had normal GTT, and 70% higher in women who were classified as gestational diabetes by the Carpenter and Coustan criteria.

(k) Spontaneous abortion

The increased likelihood of spontaneous abortion is found to correlate with high HbA1C levels.

(l) Neonatal Complications

Neonatal hypoglycaemia. The blood sugar in normal babies falls to about 50 to 60 mg/dl during the first 24 hours of life, but

in diabetic babies the level is often lower and may reach up to 25 mg/dl. Neonatal hypoglycaemia is defined as serum glucose level less than 35 to 40 mg/dl at term during the first 12 hours of life.

(m) Hyperbilirubinaemia.

Usually caused by infants immature liver function and specifically bilirubin catabolic system function , Glucuronyl transferase.

(n) Respiratory distress syndrome (RDS).

RDS secondary to pulmonary hypertension or to diabetic myocardiopathy still occur sporadically.

(o) Neonatal hypocalcaemia

This a serum total calcium concentration < 8 mg/dl in term infants and < 7 mg/dl in preterm infants. 7% of diabetic pregnancies.

(p) Polycythaemia

Possibly due to excessive production of erythropoietin in response to chronic fetal hypoxia.

(q) Neuro developmental abnormalities.

Minor cognitive disorders due to suboptimal glycemic control in pregnancy.

(r) Long-term complications.

Increased risk of obesity type 2 diabetes in children and adults who are exposed to hyperglycaemia in utero. (5)

4. TERMINOLOGY AND CLASSIFICATION

Definition of gestational diabetes mellitus used by (ACOG), (6) is any degree of glucose intolerance that either commences or is first diagnosed in pregnancy. This definition does not exclude the possibility that the diabetes may have existed but been unrecognised prior to pregnancy.(7) In 2010, the International Association of Diabetes and Pregnancy Study Group (IADPSG), An international collaborative group, recommended new terminology for GDM based on HAPO study .

Diabetes that is first recognised in pregnancy can be classified as either 'overt' or gestational'. This recognises that an increasing number of women have unrecognised type 2 diabetes at the time of conception, which is associated with a higher risk of

adverse pregnancy outcomes including congenital anomalies, as well as diabetic complications.(8)

Overt diabetes is present if any of the following are found at the first antenatal visit:

- Fasting plasma glucose ≥ 126 mg/dl (7.0 mmol/l)
- HbA1c ≥ 6.5% (on a standardized assay)
- Random plasma glucose ≥ 200mg/dl (11.1 mmol/l) plus confirmation with a fasting plasma glucose or HbA1c.

National Diabetes Data GROUP: etiologic classification of diabetes

Type I diabetes mellitus (beta-cell destruction usually leading to absolute insulin deficiency)

- Immune-mediated
- Idiopathic

Type II diabetes mellitus (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

Other specific types of diabetes

- Genetic defects of beta-cell function
- Genetic defects in insulin action
- Disease of the exocrine pancreas
- Endocrinopathies
- Drug – or chemical-induced
- Infections
- Uncommon forms of immune-mediated diabetes
- Other genetic syndromes associated with diabetes

Type III Gestational diabetes mellitus

American Diabetes Association. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care 2000; 23 (Suppl 1): S4

White's classification of diabetes during pregnancy

Gestational diabetes	-	Discovered during pregnancy, glycemia may or may not be maintained by diet alone and insulin may be required
Class A	-	Discovered before pregnancy, controlled with diet alone, any duration or age of onset
Class B	-	Onset age 20 year or older, duration less than 10 years
Class C	-	Onset age 10-19 year, duration 10-19 years
Class D	-	Onset age under 10 year, duration over 20 years, background retinopathy
Class R	-	Proliferative retinopathy or vitreous hemorrhage
Class F	-	Nephropathy with proteinuria over 500 mg/day
Class RF	-	Criteria for both classes R and F coexist
Class H	-	Arteriosclerotic heart disease clinically evident
Class T	-	Prior renal transplantation

Hare JW, White P. Gestational diabetes and the White classification. Diabetes Care 1980; 3: 394.

Unfortunately, White's classification is not ideal and should not be used alone because the number of groups is large and because patients in the same group may have completely different

prognosis. The current tendency is to classify the patients by type and then by White`s class.

5. Screening and Diagnosis

It is difficult to estimate the prevalence of GDM due to differences in diagnostic criteria as well and local and ethnic variation. With newer diagnostic thresholds, the prevalence in the general obstetric population may be up to 18 %.(8) .

Risk factors associated with GDM(9)

1. Maternal age > 30 years
2. Family H/O of type 2 DM
3. Non white ethnic – I Generation Hispanics south Asians and Middle Eastern women.
4. Obesity (> 15% of non pregnant ideal body wt / > 200 pound / >90.9kg)
5. Smoking
6. Increased weight gain in early childhood
7. Polycystic ovary syndrome
8. Previous large infant (>95thpercentile)
9. Previous unexplained still birth

10. Glycosuria
11. H/O congenital anomaly
12. H/O prematurity
13. Polyhydramnios
14. H/O GDM in previous pregnancy
15. H/O unexplained neonatal death
16. Recurrent moniliasis & UTI in present pregnancy
17. Bad obstetric history (>3 spontaneous abortions in the first/second trimester).

An increased risk of various maternal and foetal adverse outcomes have now been well – documented, although the benefits of treatment had remained controversial until recently, fuelling the debate on universal versus selective screening.

Major recent research in gestational diabetes has focused on redefining glucose thresholds for diagnosis and treatment targets, as well as more flexible approaches to treatment based on foetal parameters and expanding the treatment options available.

5.1 Screening Methods

There are several conditions that should be fulfilled in order to adopt a generalized screening method during pregnancy :

1. The condition to be screened for should have a significant impact on maternal and fetal health.
2. The screening method should have high sensitivity and specificity.
3. An effective method should be available to treat the condition and reduce its impact on the outcome of pregnancy.

Screening for gestational diabetes is performed by orally administering 50 g of glucose and measuring the venous plasma glucose 1 hour later. It is not necessary to follow a special diet before the test and it is nor necessary to be in a fasting state. Plasma glucose values should not be substituted with capillary reflectance meter glucose values. The sensitivity of the test is related to the threshold used for diagnosis and with the prevalence of the condition in the population. When 130 mg/dl is used as the threshold, the test will have a sensitivity of 90%, which decreases to 80% when the threshold is 140 mg/dl.

Some communities may have a prevalence of gestation diabetes as high as 14% and in this case the number of false positive will be small even if the lower threshold is adopted for screening.

In a study by Langer et al. (10) 555 gravidas with untreated gestational diabetes diagnosed after 37 weeks were matched with 1110 women with treated gestational diabetes and 1110 women without gestational diabetes. A composite adverse outcome was 59% for untreated, 18% for treated, and 11% for nondiabetic subjects.

The US Preventive Services Task Force (11) and the American College of Obstetricians and Gynecologists (ACOG) recommend selective screening of high-risk women. However, most obstetrical practices find it impractical to select patients at high risk, and generalized screening is predominant.

Risk assessment and timing of screening for gestational diabetes

Low risk

All of the following:

- Member of an ethnic group with a low prevalence of GDM
- No known diabetes in first-degree relatives
- Age < 25 years
- Weight normal before pregnancy
- Weight normal at birth

} Blood glucose screening not routinely required

- No history of abnormal glucose metabolism

Average risk

One or more of the following:

- Member of an ethnic group with a high Prevalence of GDM
- Diabetes in a first-degree relative
- Age ≥ 25 years
- Overweight before pregnancy
- Weight high at birth

} Blood glucose testing at 24-28 weeks (one-or two- step procedure)

High risk

- Marked obesity
- Strong family history of type II DM
- Previous history of GDM, impaired glucose Metabolism or glucosuria

} Perform glucose testing as soon as feasible

According to the 1997 recommendations, screening and diagnosis were undertaken as a 'two-step' approach. If the screening test, the glucose challenge test, is positive, that is the plasma glucose level is ≥ 140 mg/dl, the diagnostic test, 3-hour 100 g GTT is recommended. Using the cut-off a 140 mg/dl, about 80% of gestational diabetics can be detected and 15% patient will need to undergo GTT.

5.2 Targeted or Universal Screening?

The American Diabetes Association (ADA) recommends that women are low risk and need not undergo routine screening if they meet all of the following criteria: age < 25years, normal weight, not of a high-risk ethnic group, no family history of diabetes, no personal history of abnormal glucose metabolism or

poor obstetric outcome. It also recommends early screening for GDM (in the first trimester) if there is a history of severe obesity, previous GDM or large for gestational age (LGA) infant, glycosuria, polycystic ovarian syndrome, or a family history of type 2 diabetes, with re-testing at 24-28 weeks gestation if the initial screening is negative. (14) However, a study that attempted to apply these criteria found that only 10% of women actually met all of these criteria and thus avoided the need for screening. (15) Therefore, in the interest of simplicity, many other organizations recommend universal screening.

5.3 GDM detection: Screening vs. Diagnostic Testing

Two major approachers to screening for GDM have traditionally been considered. The two-step approach starts with a 50 g glucose challenge test (GCT) as a screening test, followed by a 75 g or 100 g oral glucose tolerance test if the GCT is positive. A one step approach has been endorsed by the DIPSI, whereby the initial test is a diagnostic 75 g, 2-hour oral glucose tolerance test (OGTT).

Recommends the one-step diagnostic OGTT between 24 and 28 weeks gestation, at detecting both overt diabetes in early pregnancy, as well as true gestational diabetes at a later gestation.

6. Criterions

Diagnosis of GDM by 100-g 3-hr OGTT by Carpenter and Coustan (1982)(12)

- Fasting 95 mg/dl
- 1 hour 180 mg/dl
- 2-hour 155 mg/dl
- 3-hour 140 mg/dl

According to the new recommendations, all women not known to have diabetes earlier should undergo a 75-g OGTT at 24-28 weeks of gestation. A fasting blood sample is drawn, following which the woman is instructed to drink a solution of 75 gm glucose dissolved in a glass of about 300 ml of water over a period of 5 – 10 minutes. Some lemon juice can be added to the glucose water to prevent nausea and vomiting that so often follows the rapid ingestion of so large a quantity of glucose on an empty stomach. Thereafter, plasma glucose levels are estimated after 1 hour and 2 hours, which means that total three blood samples are taken. Gestational diabetes is diagnosed if any one of the three values is met or exceeded.

Diagnosis of GDM by 75-g OGTT: IADPSG guidelines(2011) (13) OGTT performed in the morning after overnight fast of at least 8 hours

Fasting $\geq$ 92 mg/dl (5.1 mmol/l)

1 hour $\geq$ 180 mg/dl (10.0 mmol/l)

2 hours $\geq$ 153 mg/dl (8.5 mmol/l)

Diagnosis of GDM is made when any one value is exceeded

The initial criteria used for diagnosis of GDM were established in the 1960s,(16) and have undergone only slight modifications since then. The World Health Organization (WHO) still uses the same diagnostic criteria for impaired glucose tolerance in pregnancy as in non-pregnant individuals,(17) although acknowledging more recently that this includes both impaired glucose tolerance and diabetes.(18)

Criteria followed by WHO and IADPG for a positive 75 g OGTT in pregnancy are described below:

Criteria for a Positive 75 g OGTT in Pregnancy			
	Fasting plasma glucose	**1-hour plasma glucose**	**2-hour plasma glucose**
World Health Organisation	≥ 125 mg/dl ≥ 6.9 mmol/l		≥ 140 mg/dl ≥ 7.8 mmol/l
IADPSG and American Diabetes Association	≥ 92 mg/dl ≥ 5.1 mmol/l	≥ 180 mg/dl ≥ 10.0 mmol/l	≥ 153 mg/dl ≥ 8.5 mmol/l

7. LITERATURE REVIEW

The DIPSI (Diabetes in pregnancy study group India) recruited about 1463 pregnant women between April 2009- February 2010 and using the criteria following the 75gm glucose 2 hr test . Statistical analysis was done using chi-square test and independent-t test . The mean age was 23.60 ± 3.32yrs. The BMI was 21.5 ± 4.06 kg/m2 . The average age of gestation was 27.9 ± 5.56 weeks. This study showed 196 women which is 13.4% to have GDM and the remaining had normal values . They studied birth weight and followed up the neonate following treatment with either insulin or diet regulation and found out that macrosomia was seen in 9.9% GDM mothers and 9.8% in the normal blood glucose level mothers and there was no significant statistical difference between the GDM and normal blood glucose value mothers (p=1.000). However this DIPSI study did not have a control group which had untreated antenatal mothers with 2h PG $\geq$ 7.8 mmol/L (19).

Advantages of DIPSI

- Fasting status not required
- Does not alter her routine activities
- Both screening and diagnostic

DIPSI CRITERIA	IMPAIRED GLU TOL(IGT)	GDM	OVERT DM
2HR AFTER 75GM	120-139	140-199	>200

Normal < 119 mg/dl

International Association of The Diabetes and Pregnancy Study Group (IADPSG) Disadvantages

- dropout rate is very high due to the necessity of fasting blood sample
- FPG values do not reflect the 2hr 75gm GCT which is supposedly the diagnostic test for GDM.
- Asian Indian ethinicity have high insulin resistance and so their 2hr PG is higher than Caucasians.

Hence FPG is not an appropriate test for diagnosis of GDM(20). As a result FPG > 5.1mmol/L will miss 76% of GDM by the WHO criteria(21)

WHO Criteria :

WHO CRITERIA	IMPAIRED GLU TOL(IGT)	GDM	OVERT DM
FBS	100-109	110-125	>126
2HR PPBS	120-129	140-199	>200

Normal Values

FBS- <99

2HR PPBS- <120

In a study which enrolled 1463 pregnant women who underwent IADPSG and DIPSI criteria. The objective of this study was to find out if DIPSI could diagnose GDM against the IADPSG. The prevalence of GDM with DIPSI was 13.4% (n=196) and IADPSG was 14.6% (n=214) and concluded that there was no statistical significance (P=0.21) between the two test and therby implied a close agreement between the two tests.

In another Pilot study done by Vijayalakshmi et al at PSIMS & RF in Andra Pradesh which was to assess the effectiveness of DIPSI in diagnosing GDM. This study enrolled 200 antenatal women who underwent DIPSI followed by the ADA criteria 3 days later out of the 200 women 22(11%) had abnormal DIPSI. And out of the 22 only 5 (2.5%) had abnormal ADA. As a result 17 women were wrongly caterogised had GDM by DIPSI. Therby a prevalence of 2.5% was detected . The sensitivity being 100% and Specificity 89% and false positives 1.6% (22) .

Lee at al observed glucose tolerance decreases in the afternoon and evening as detected by the intravenous and oral GTT reduction in the insulin sensitivity and β-cell responsiveness are responsible for this glucose tolerance deterioration later in the day.

The hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study (23). This was a seven – year international epidemiological study of 23,325 pregnant women at 15 centers in nine countries that comprised the HAPO Study Cooperative Research Group (2008). The investigation was designed to determine the association of various levels of glucose intolerance during the third trimester

with adverse infant outcomes in women with gestational diabetes. Between 24 and 32 weeks, the general population of pregnant women underwent 75-g oral glucose tolerance testing after overnight fasting. Blood glucose levels were measured pretest fasting and again, 1 and 2 hours after glucose ingestion. Caregivers were blinded to results except for women whose glucose levels exceeded values that required treatment and removal from the study. Values at each of three time posts were stratified into seven categories and analyzed for birthweight > 90^{th} percentile (LGA), primary cesarean delivery, clinical neonatal hypoglycemia, and cordserum C-peptide levels > 90^{th} percentile. Odds of each outcome were calculated using the lowest category – for example, fasting plasma glucose < 75 mg/dl – as the referent group. Their findings in general supported the supposition that increasing plasma glucose levels at each epoch were associated with increasing adverse outcomes.

8. MANAGEMENT

8.1 Nutritional Treatment – Life style intervention

8.1(a) Total daily caloric intake

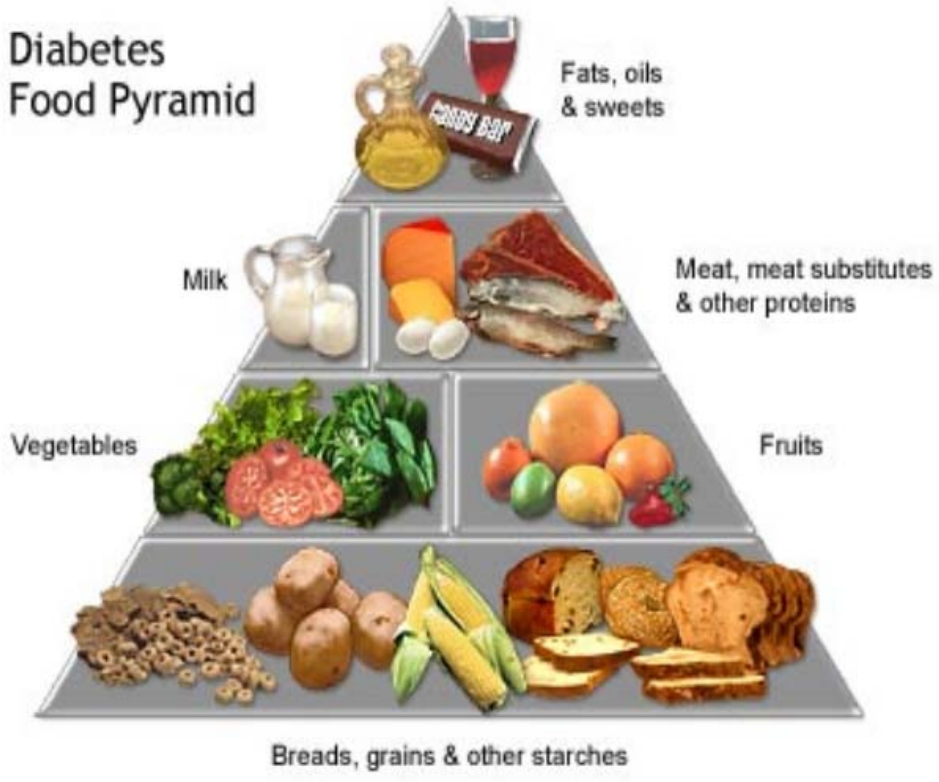
The first step in the meal planning for GDM or other pregnant diabetics is to calculate the optimal total daily caloric intake. Calculation of the total daily caloric intake is based on the number of calories necessary to maintain 1 kg of body weight, which is 30 kcal for the average normal-weight women (80-120% ideal body weight), 35-40 kcal for women who are underweight (less than 80% ideal body weight), 25 kcal for overweight women (121-150% ideal body weight), and 12 kcal/kg for morbidly obese women (more than 150% ideal body weight). This number is multiplied by the body weight in kilograms to obtain the total number of calories that the patient should consume during a 24-hour period.

For the majority of women with GDM the optimal total daily caloric intake will be between 2000 and 2500 cal/day.

In the third trimester of pregnancy, when the caloric needs of the mother are increased, it is prudent to add 300 kcal to cover the additional caloric needs of the pregnancy. The total caloric

intake is split into three meals and one to three snacks depending on the patient's habits.

The daily caloric allowance should be distributed among the different food groups in such a way that approximately 40-50% of the calories come from complex carbohydrates. The carbohydrate content of the diet should be distributed as 10-15% at breakfast, 20-30% at lunch, and 30-40% at dinner. Snacks should have 0-10% of the total carbohydrates. The rest of the caloric intake comes from fat (30-40%), predominantly unsaturated and protein (24).



(b) Glycemic index

Different carbohydrate products have different impacts on blood glucose levels. To account for these differences the concept of the glycemic index has been developed. The glycemic index of a particular carbohydrate is the blood glucose response to that product in a given period of time compared to the blood glucose of a similar amount of carbohydrate in a standard food, usually white bread. Several studies in nonpregnant populations have demonstrated that ingestion of carbohydrates with low glycemic index results in better blood glucose control and better serum lipid profiles.

(c) Exercise

Exercise improves glycaemic control through improving insulin sensitivity, Particularly at the level of skeletal muscle. Even very light exercise (walking 2 .52 km in 1 hour) done after eating has been shown to significantly reduce the 1-hour postprandial blood glucose. (25)

The treatment was safe, with no cases of post-exercise hypoglycaemia, and no differences in caesarean section rate, macrosomiaor preterm delivery.

8.2 Glucose Self-Monitoring

Monitoring of home blood glucose is necessary in order to identify women at increased risk of adverse perinatal outcomes and to determine the need for intensification of therapy. It has been shown to have a number of benefits for the mother and the foetus. (26)

8.2 (a) Fasting Blood Glucose

The HAPO study demonstrated an increase in adverse perinatal outcomes with elevation of the fasting glucose alone on OGTT.(27) An earlier study of women with treated GDM had showed a correlation between increasing levels of fasting glucose > 95 mg/dl and adverse neonatal outcome.

(b) Post-Prandial Versus Pre-Prandial Blood Glucose

Post-prandial blood glucose monitoring has been suggested to be superior to pre-prandial in GDM. Post-prandial glucose monitoring may be performed either one or two hours after a meal, with no clear benefit for either approach at present.(28)

8.3 Glucose targets

The current therapeutic targets recommended by the ADA are: (29)

- Fasting blood glucose level ≤ 95 mg/dl (5.3 mmol/l)
- 1-hour post-prandial glucose level ≤ 140 mg/dl (7.8 mmol/l)
- 2-hour post-prandial glucose level ≤ 120 mg/dl (6.7 mmol/l)

9. PHARMACOTHERAPY

9.1 Insulin

Insulin has been a standard treatment for many years for gestational diabetes. The most common indication for insulin treatment in GDM women is persistent elevation of the FCG value. This reveals altered hepatic neoglucogenesis not modified by glyburide.

9.1(a) Types of insulin.

The types of insulin used in pregnancy are as follows:

- Rapid-acting insulin analogues – lispro and as-part
- Short-acting human insulin – regular insulin
- Intermediate-acting insulin – NPH (isophane)

(b) Insulin and their action

Type of Insulin	Generic Name	Onset	Peak	Duration
Rapid-acting	Lispro, Aspart	15 min	30-90 min	3-5 hrs
Short-acting	Regular	30-60 min	2-4 hrs	5-8 hrs
Intermediate-acting	NPH	1-3 hrs	8 hrs	12-16 hrs

However, recent research has focused on the safety of the newer insulin analogs in pregnancy. These are an attractive option due to more convenient timing of administration (aspart, lispro) and a lower risk of hypoglycaemia (glargine).

In the only prospective observational study of glargine compared to NPH insulin in women with gestational and pre-gestational diabetes, glargine was associated with a decreased risk of mild and frequent hypoglycaemia compared to NPH, and was not associated with any increase in adverse outcomes. In fact, there was a statistically significant reduction in foetal anomalies, foetal death and admission to neonatal ICU in the glargine group, although the overall numbers were very small.(30)

The majority will respond to continuation of treatment with glyburide plus a single injection of glargine insulin (Lantus) in the morning or NPH at bed time. The rationale for using a combination of oral hypoglycemic agent plus insulin is that insulin can suppress hepatic neoglucogenesis, which is the primary cause of elevated fasting hypoglycemia.(31) The use of insulin glargine results in less episodes of nocturnal hypoglycemia than that of NPH. The optimal time for insulin administration depends of the type of insulin being used. NPH seems to be more effective at bed time while insulin glargine provides better control when given in the morning. If the PG values remain elevated when using insulin glargine, the dose may be increased by 5 U every 5 days until adequate blood glucose control is obtained.

(c) Oral Hypoglycaemic Agents

Although insulin has been the traditional first-line treatment for GDM when nutritional therapy fails, the use of oral hypoglycaemic agents in the treatment of GDM is appealing to patients and providers due to ease of administration.

c(i) Safety of OHA's

Despite placental transfer, glyburide appears to be safe for the foetus up to a materal dose of 10mg BD.(32). One study of 332 infants born to mothers with type 2 diabetes found that there was no difference in rates of anomalies with different forms of diabetic treatment in the first trimester (diet, insulin or sulfonylurea) (33).

In an RCT by Langer et al, 400 women were assigned to receive either glyburide or insulin when intensification of treatment was required for GDM.(34).

No significant differences were found in terms of incidence of LGA infants, macrosomia, lung complications, neonatal hypoglycaemia, admission to neonatal ICU or congenital anomalies.

(d) Metformin

Metformin in Gestational Diabetes (MiG) trial assigned women with GDM at to either metformin, with insulin added if required, or insulin alone.(35) The only significant differences in individual components of the primary outcome was increased neonatal hypoglycaemia (< 28.8mg/dl, 1.6mmol/l) in the insulin

group (8.1% vs. 3.3%), and an increase in preterm birth < 37 weeks in the metformin group (12.1% vs. 7.6%).

Metformin therapy was associated with a number of benefits, including a reduced dose of insulin required (42units vs. 50units per day in those on insulin), and increased acceptability (76.6% said they would choose to receive this treatment again, vs 27.2% in the insulin group). Patients taking metformin gained less weight from enrolment to 36-37 weeks gestation (0.4kg vs. 2kg), and also lost more weight from enrolment to the postpartum visit. Conclusion of MiG trial was that metformin, either alone or in combination with insulin, is safe and effective as a treatment for gestational diabetes, with benefits including patient acceptability and reduced weight gain.

10. Adjusting Treatment Based on Foetal Ultrasound Parametres

The rationale for this approach is that even with strict control of GDM there is still an increased risk of macrosomia in some infants,(36) whilst some foetuses may be at risk of growth restriction in this situation due to excessively tight maternal glucose control.(37)

- AC $\geq$ 75th percentile: fasting < 80 mg/dl (4.4 mmol/l) and post-prandial < 100 mg/dl (5.5 mmol/l)
- AC < 75th percentile: fasting < 100 mg/dl (5.5 mmol/l) and post-prandial < 140 mg/dl (7.8 mmol/l)

This modified treatment resulted in a significant reduction in the percentage of LGA infants (7.9 vs. 17.9%), SGA infants (6.0 vs. 9.0%), and macrosomia (3.3 vs. 11%).

11.Foetal Surveillance and Timing of Delivery

They noted that the only deaths in normally formed infants occurred when there was clinical evidence of foetal macrosomia, polyhydramnios or poor metabolic control. Consequently in their absence, this group of experienced clinicians allowed the otherwise uncomplicated pregnancies to go to full term (40 completed weeks of gestation).(38)

Gabbe and colleagues recommended that in uncomplicated GDM Pregnancies, CTG monitoring should be commenced after 40 weeks' gestation whilst awaiting spontaneous onset of labour.(39)

A Cochrane review(40) published in 2001 found that there was only one randomized controlled trial(41) comparing planned elective delivery at 38 weeks' gestation vs. expectant or awaiting the onset of spontaneous labour up to 42 weeks' gestation, with twice weekly CTG and amniotic fluid volume surveillance. The review concluded that induction at 38 weeks did not result in an increase in caesarean section RR 0.81 (95% CI 0.52-1.26). However, the risk of macrosomia (birthweight ≥ 4000 g) was lessened in the elective delivery group RR 0.56 (95% CI 0.32-

0.98) and there were three cases of mild shoulder dystocia in the expectant group.

12. MODE OF DELIVERY

The major concern regarding vaginal delivery in omen with gestational diabetes is the potential risk of shoulder dystocia and in particular resultant brachial plexus palsy. What ultimately determines if the foetal shoulders will pass readily through the maternal pelvis is the dynamic interaction between the maternal pelvic girdle, the strength of the uterine contractions and the mother's expulsive efforts and the foetal diameters, none of which can be reliably measured and/or predicted. Mactrosomic (i.e., birthweight > 4000 g).

13. POSTPARTUM

There is a sharp fall in the patient`s insulin requirements immediately after delivery. For insulin dependent diabetics, the usual practice is to start them on about half the dose of insulin before delivery, or the pre-pregnancy dose. If the patients has delivered by caesarean section, rapid-acting insulin may be used to treat glucose levels greater than 140-150 mg/dl by multiple dose injections or continuous insulin infusion until she is orally allowed.

Gestational diabetics controlled on diet alone can revert to their normal diet postpartum, and those who needed insulin during pregnancy usually do not require it any longer.

All gestational diabetics should be advised to have fasting blood sugar tested at 6 weeks, and annually thereafter.(42) They should be counselled regarding diet, exercise and weight reduction which can reduce their chances or delay developing type 2 diabetes later.

14. BREASTFEEDING

Early breastfeeding, within 30 minutes of birth, and every 2-3 hours, also helps in reducing the risk of neonatal hypoglycaemia.

Women with pre-existing diabetes can resume or continue to take metformin and glibenclamide while breastfeeding but other oral hypoglycaemic agents should be avoided.

15. CONTRACEPTION

Copper intrauterine devices, battier methods, and natural family planning methods can be used without restriction in all diabetics (type 1 and 2). Though there has been a concern regarding an increased risk of infection and pelvic inflammatory disease with the use of intrauterine decices in diabetics, there is no evidence to support such fears. The World Health Organization advises unrestricted use of copper intrauterine devices in all types of diabetics.(43)

Women with diabetes mellitus and nephropathy, retinopathy, neuropathy, or other vascular disease are not advised to use progestogenonly injectables, COCs, combined contraceptive patch and the vaginal ring.(44).

A permanent method of contraception like tubal ligation can be offered but should be undertaken with caution in those with vasculopathy and hypertension.

16. PREVENTION OF GESTATIONAL DIABETES

1 .Pre- conceptional counseling

- The risk of future DM
- Life Style advice regarding diet and exercise to avoid obesity

Weight loss prior to pregnancy would be predicted to reduce the risk of Developing GDM. Women who exercised the most pre-pregnancy (by self-report) had a 55% lower risk of developing GDM, and the risk of GDM was also reduced by 24% in women with the most exercise in early pregnancy.(51)

2. Metformin before and during pregnancy in women with PCOS

According Glueck et al and and Jakubowicz et al, there is 10 fold reduction in incidence of GDM, in first trimester miscarriage.

17. INDIAN EXPERIENCE OF DIABETES COMPLICATING PREGNANCY

With improvement in antenatal care and routine screening of all pregnant women for carbohydrate intolerance, an increasing number of cases of diabetes are being detected of all cases of diabetes complicating pregnancy, the majority (about 90%) are cases of gestation diabetes. The incidence quoted by Indian authors are presented in Table below. In summary, the incidence of gestation diabetes is about 3-5% in India.

Incidence of diabetes complicating pregnancy in India (50)

Authors	Year	Incidence (%)
Goel and Bathla	1999	2-5
Ganguli et al.	1995	0.25
Maheshwari et al.	1989	4.9
Bhattacharya et al.	2001	3.0
Kumar et al.	1993	5.5
Sridhar and Nagamani	2003	12.7
National Diabetes Data Group	2003	4.5

In studies on the effects of diabetes in pregnancy, the following pertinent observations were reported.

- The incidence of GDM (9.84%) was high in women undergoing spontaneous Abortions (Zargar et al., 1995),(45) particularly in those with poor glycemic control.
- The incidence of GDM varies widely even within the Same metropolitan area (Ramachandran and Snehalatha, 1998).(46)
- Excessive weight gain was observed in 32% of women with GDM as compared to 1.7% in controls (Jindal et al., 2001).(47).
- Fetal macrosomia was observed in 32% of GDM women as compared to 6.8% in controls (Jindal et al., 2001).
- Incidence of pregnancy-induced hypertension was 48% in GDM group as compared to 18.8% in controls.
- Incidence of hydramnios reported was 28% in the GDM group as compared to 4.3% in controls (Jindal et al., 2001).

- Candidal vulvovaginitis was reported in 4% in the GDM group as compared to 1.3% in controls (Jindal et al., 2001).
- Incidence of intrauterine fetal deaths was 12% in the GDM group as compared to 1.7% in controls (Jindal et al., 2001).
- Incidence of fetal malpresentations was 16% in GDM group as compared to 6% in controls (Jindal et al., 2001).
- Cesarean section was required in 44 % of GDM group as against 13.3% in controls (Jindal et al., 2001).
- Postpartum complications are also much higher in the GDM group (Buckshee and Rohatgi, 2003).(48)
- Maternal mortality is 10 times higher in GDM patients (Buckshee and Rohatgi, 2003).
- Perinatal morbidity increases in GDM patients (Agarwal et al., 1993; Ramachandran and Snehalatha, 1998)
- Strict glycemic control of GDM improves obstetric outcome significantly. The glycemic control should ideally precede onset of pregnancy.

- Prescribing pyridoxine 40 mg twice daily in GDM patients helps to reduce insulin requirements to control glycemic control (Vijayakumar et al., 1991; Arjun, 1998).(49)

Aim and Objectives

AIM AND OBJECTIVES

To evaluate if a single blood test report after a 2hr oral glucose load irrespective of the last meal according to the DIPSI criteria can be used as a diagnostic test for Gestational diabetes mellitus and compare the values with that of the WHO recommendation which is a two step process which consists of a FBS followed by 2hrs later 75gms oral glucose test blood test.

Materials and Methods

MATERIALS AND METHODS

The study was conducted in the department of Obstetrics and Gynaecology, PSG Hospitals, Coimbatore from September 2013 – 2014.

The study period was 10 months.

STUDY DESIGN

Prospective Cross Sectional Study

STUDY POPULATION

The study group consisted of 108 women attending ante – natal clinic in the Department of Obstetrics & Gynaecology between 24 – 28 weeks of Gestation attending the OP Department in PSG Hospitals .

SELECTION CRITERIA

All antenatal women from 24 – 28 weeks of gestation willing to participate in the study .

EXCLUSION CRITERIA

Antenatal patients on steroids for autoimmune disorders / obstructive lung disease.

Overt Diabetes Mellitus

Type 1 – juvenile Diabetes Mellitus

<24 weeks and > 28 weeks of gestation

Patients who had only either of the two testing done.

METHODOLOGY

The study was initiated after obtaining approval of the ethics committee in PSG IMSR.

The patients selected were according to the inclusion criteria between 24-28 weeks of gestation and oral and written consent was obtained.

Patients who were not complient during the study or vomited after ingestion of 75gm glucose or did not turn up for both the studies were excluded.

Basic assessment of their risk factors was already done in the first antenatal visit. If not their history along with a detailed family history, presence of GDM in first pregnancy in case of multigravidas was also noted.

Their height and weight was measured. Weight was noted at the time of first visit.

Gestational age was noted for both the tests.

Other women who were already overt Diabetes or type I Diabetes,and patients on other medical problems on steroids were excluded from the study.

About 108 antenatal mothers aged 18-35yrs with gestational age 24-28 weeks of pregnancy who attended the OPD in PSGIMSR between September 2013 - September 2014 was taken who are eligible for the study were explained about the study after excluding other women who were not eligible for the study.

Timing of the last meal was noted. Non compliance and problems during the tests were also noted.

All the patients were instructed not to have meals after the 75gm glucose ingestion.

All the patients were asked to follow unrestricted carbohydrate diet and not to change the diet pattern inbetween the two tests.

All the antenatal women between 24-28 weeks of gestation were on Test 1- irrespective of the last meal given 75gms oral glucose mixed in 150 ml of water and blood test taken 2 hrs later according to

the DIPSI criteria. If the patient experienced nausea during the drinking procedure a pinch of fresh lime was added. If she had vomited after glucose ingestion then the testing is done at the further time of the day or asked to come on the following day for re-testing and the same procedure is followed. The patient is requested not to have meals in between and venous blood was collected 2 hrs later. Two ml of venous blood was collected in sterile vacutainers containing lithium heparin. These samples were centrifuged and analysed for glucose by glucose oxidase peroxidase(GOD-POD) method using commercial Randox kits and run on an automated Randox Daytonal clinical chemistry analyser within 1 hr. After standardization the internal quality control samples were run.

Glucose oxidase (GOD) converts glucose to gluconic acid. Hydrogen peroxide formed in this reaction, in the presence of peroxidase (POD), oxidatively couples with 4-aminoantipyrine and phenol to produce red quinoneimine dye. This dye has absorbance maximum at 505 nm (500-550nm).The intensity of the color complex is directly proportional to the concentration of glucose in the specimen.

After the test 1 all patients were asked to come back within 3 to 7 days with 8 hrs fasting status for the test 2.

Test 2- after 3 to 7 days of unrestricted carbohydrate diet the same patients who underwent DIPSI testing came in fasting status .First a fasting venous sample was taken . Following which 75gm oral glucose was given to the patient and 2hr later another venous sample was taken.The results were processed just as mentioned above and analysed for blood glucose.

Both the tests were analysed together and categorized as Normal, Impaired or Gesational Diabetes mellitus according to the DIPSI or WHO criteria.

And the diet counseling was done only prior to test 1,no diet counseling was given inbetween the tests. This was to ensure both the tests were done unbiased.

WHO CRITERIA	IMP GLUCOSE TOL(IGT)	GDM	OVERT DM
FBS	100-109	110-125	>126
2HR PPBS	120-129	140-199	>200

Normal Values

FBS- <99

2HR PPBS- <120

DIPSI CRITERIA	IMP GLUCOSE TOL(IGT)	GDM	OVERT DM
2HR AFTER 75GM	120-139	140-199	>200

Normal <119 MG/DL

Both the criterias were analysed and the results were tabulated. Women diagnosed as GDM were managed appropriately.

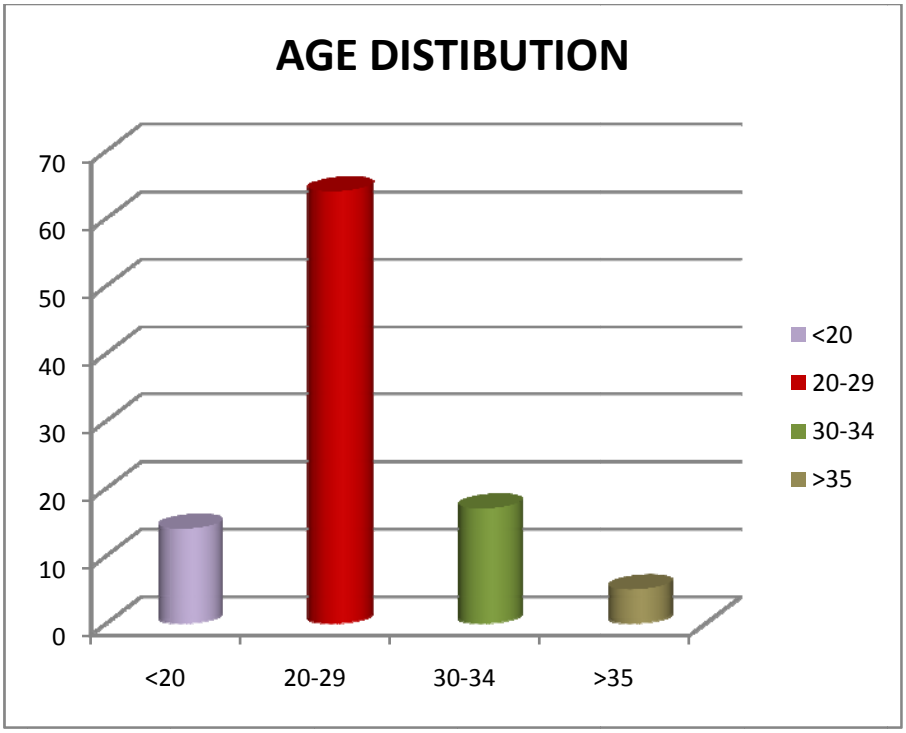
Results and Analysis

RESULTS AND ANALYSIS

1.BASELINE CHARECTERISTICS OF THE STUDY GROUP

Table-1

Age	NUMBER OF PATIENTS
<20	14
20-29	64
30-34	17
>35	5



The mean age of our patients were 26 yrs.

There were 14 patients in the underage category

(less than 20 yrs).

64 patients were between 20-19yrs,

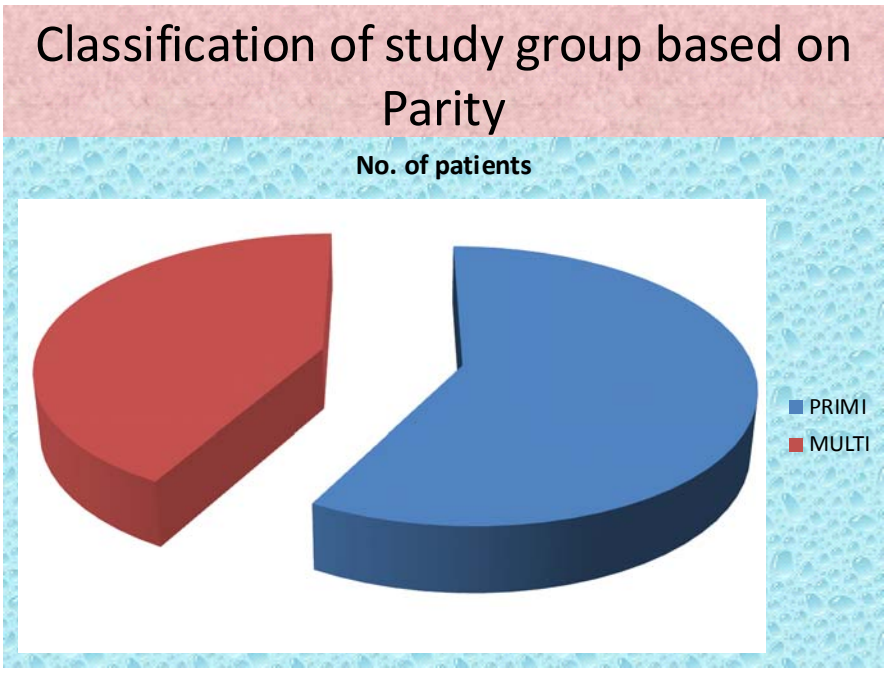
17 patients between 30-34 yrs,

5 patients greater than 35 yrs.

2. COMPARISON BASED ON PARITY

Table-2

Parity	Number of patients
Primi gravida	52
Multi gravida	48



Total number of patients - 100

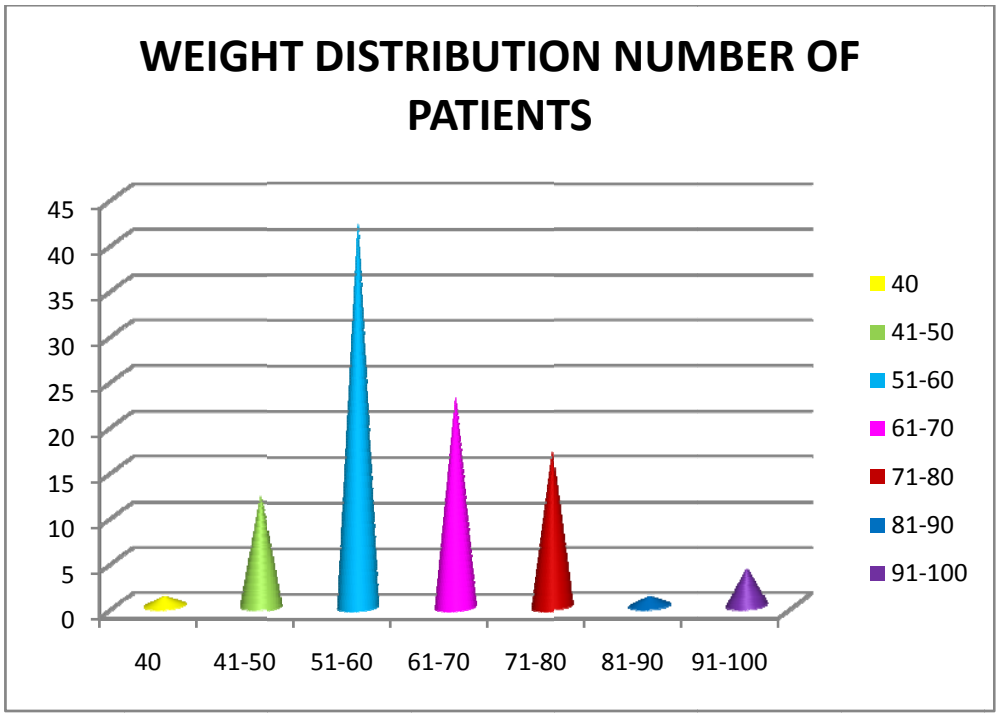
Number of primis – 52

Number of multis – 48

3. WEIGHT DISTRIBUTION

Table 3

Weight (in Kg)	NUMBER OF PATIENTS
40	1
41-50	12
51-60	42
61-70	23
71-80	17
81-90	1
91-100	4



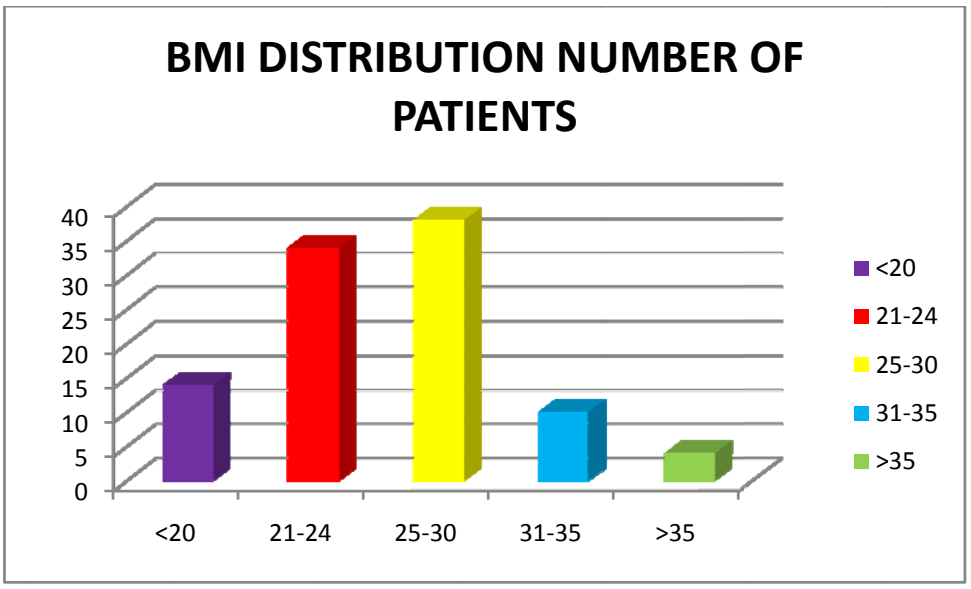
Out of the 100 patients recruited in this study there was 1 patient belonging to the underweight category(less than 40 kg), 41 to 50 kgs- 12 patients, 51 to 60 kgs- 42 patients, 61 to 70 kgs- 23 patients, 71to 80 kgs-17 patients,81 to 90kgs-1 patient,91 to 100- 4 patients.

The average weight was 52 kgs.

4. BMI DISTRIBUTION

Table - 4

BMI (Kg /m^2)	NUMBER OF PATIENTS
<20	14
21-24	34
25-30	38
31-35	10
>35	4



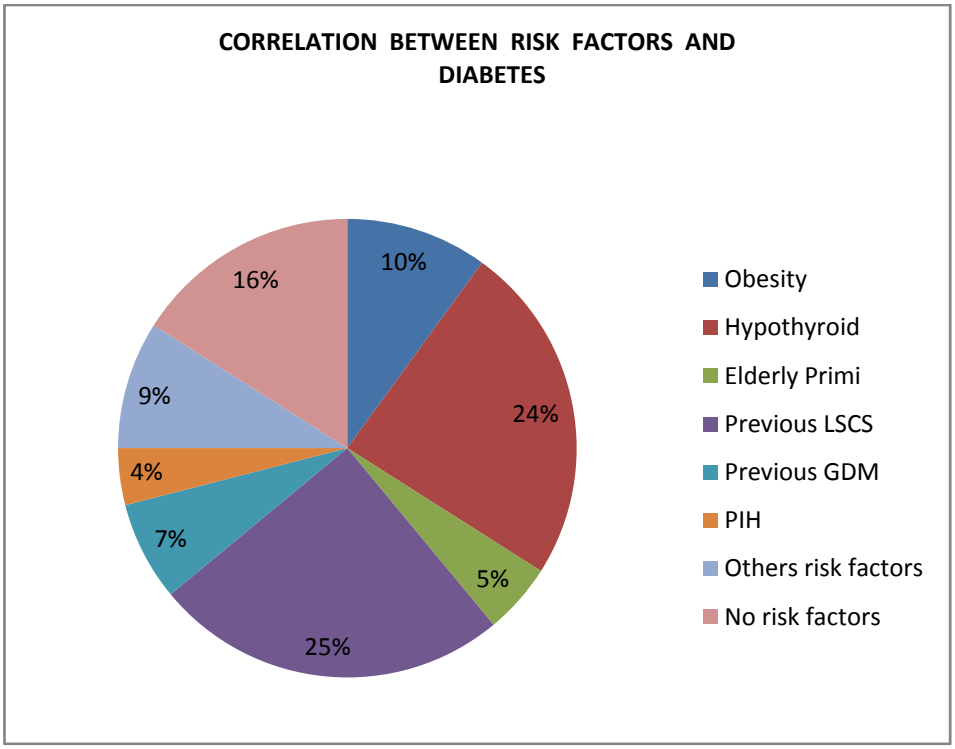
The average BMI was 25 kg/m2.

Out of which 14 patients belonged to the low BMI group of less than 20, 34 patients with BMI 21-24 kg/m2, 38 patients with BMI 25-30 kg/m2, 10 patients with BMI 31-35 kg/m2,and 4 patients BMI greater than 35 kg/m2. In the high BMI category >30kg/m2 there were 14 patients out of which 4 patients were positive in both DIPSI & WHO criteria. 2 patients had GDM according to WHO and 3 patients had GDM according to DIPSI criteria. 4 patients had normal blood sugar in both the criteria's.

5. CORRELATION BETWEEN RISK FACTORS AND DIABETES:

Table 5

RISK FACTORS	Percentage
Obesity	10%
Hypothyroid	24%
Elderly Primi	5%
Previous LSCS	25%
Previous GDM	7%
PIH	4%
Others risk factors	9
No risk factors	16



Of the risk factors , Hypothyroid was found in 24 patients and previous LSCS 25 , Obesity - 10%, followed by previous pregnancy GDM - 7, elderly primi 5, hypertensive disorders in pregnancy 4%.

Others 25

Of which Anaemia 2, Rh issoimunisation 1, chronic hypertension 1, Bad obstetric history 3, asthma (not on steroids) 2 .

Of which 16 patients had no risk factors.

6. GESTATIONAL AGE AND BLOOD SUGAR VALUES OF THE STUDY GROUP

Table 6

CRITERIA	**BLOOD SUGAR(mgs/dl)** **(Mean ± SD)**
WHO s criteria	Fasting - 87.63 ± 8.129 Postprandial - 109.71±20.57
Dipsi criteria	109.65 ± 17.97

Mean FBS & PPBS in WHO = 87mg/dl & 109 mg/dl respectively.

Mean blood sugar level in DIPSI = 109 mg/dl.

7. TESTS

Table -7

WHO CRITERIA	IMP GLUCOSE TOL(IGT)	GDM	OVERT DM
FBS	100-109	110-125	>126
2HR PPBS	120-129	140-199	>200

Normal Values

FBS- <99

2HR PPBS- <120

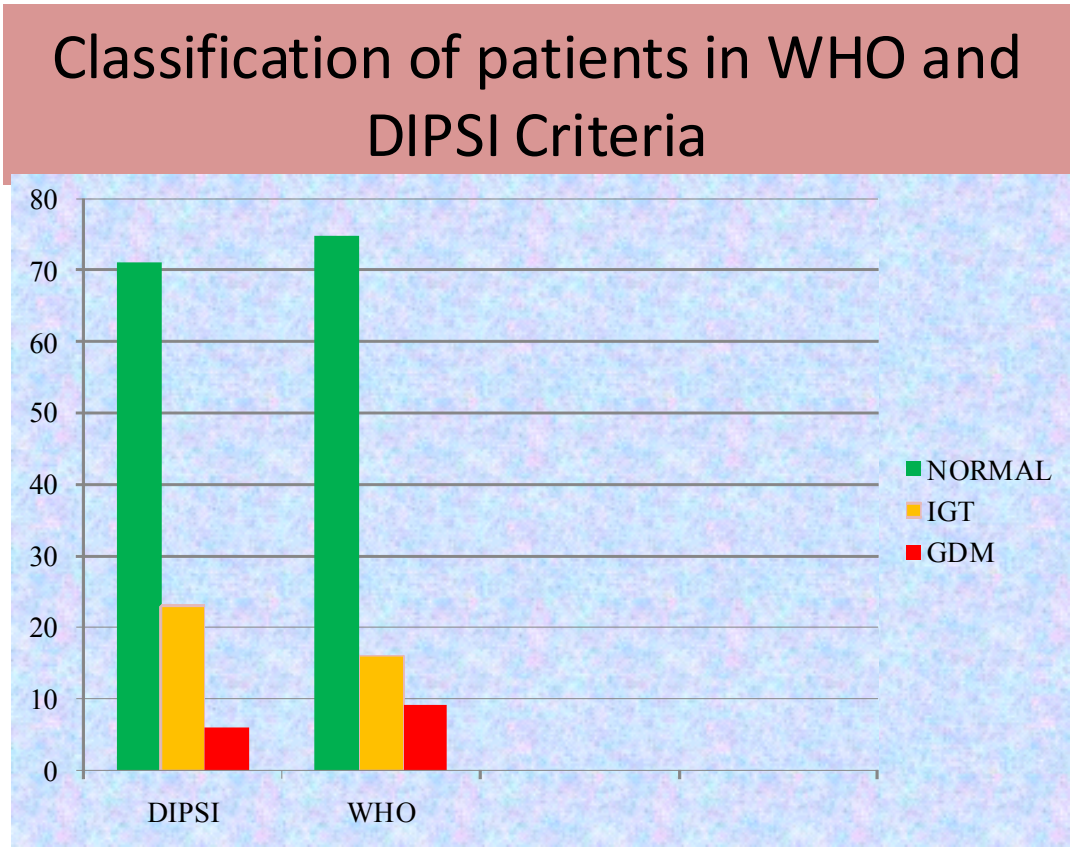
DIPSI CRITERIA	IMP GLUCOSE TOL(IGT)	GDM	OVERT DM
2HR AFTER 75GM	120-139	140-199	>200

Normal <119 MG/DL

8. CLASSIFICATION OF STUDY GROUP BASED ON THEIR BLOOD GLUCOSE LEVELS

Table-8

Classification	WHO CRITERIA	DIPSI CRITERIA
Normal	75	71
Impaired Glucose Tolerance	16	23
Gestational Diabetes Mellitus	9	6



WHO criteria :

Total number of patients with normal blood glucose = 75

Number of patients with Impaired glucose tolerance = 16

Number of patients with GDM = 9

DIPSI criteria :

Total number of patients with normal blood glucose = 71

Number of patients with Impaired glucose tolerance = 23

Number of patients with GDM = 6.

9. CALCULATING SPECIFICITY ,SENSITIVITY , AND PREDICTIVE VALUES FOR DIPSI CRITERIA.

TABLE - 9

Dipsi	**WHO's test**		
	Disease (+)	**Disease(-)**	
Positive	True positive(a) (11)	False positive(b) (19)	30 (a+b)
Negative	False negative (c) (15)	True negative(d) (55)	70(c+d)
Total	**26 (a+c)**	**74 (b+d)**	**100 (a+b+c+d)**

True positives (a) = patients tested positive in both groups DIPSI & WHO

False positives (b) = patients tested negative in WHO & positive in DIPSI

False negatives(c)= patients tested positive in WHO & negative in DIPSI

True negatives (d)= patients tested negative in both groups DIPSI & WHO

Sensitivity of Dipsi Criteria = a / a + b X 100

11 /26 X 100 = **42.3%**

Specificity of Dipsi Criteria = d / b + d X 100

55 / 74 X 100 = **74.32 %**

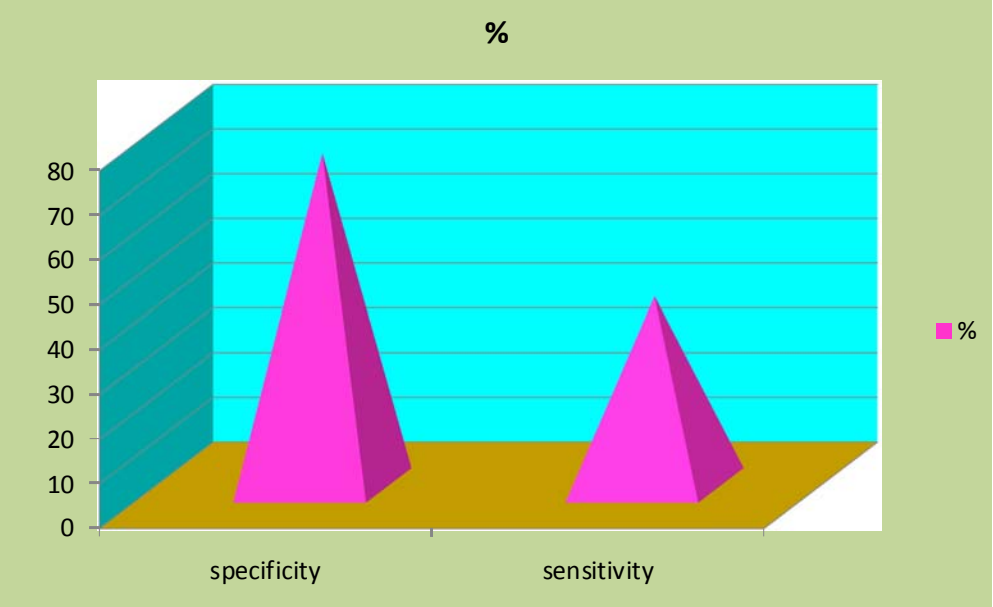
Positive predictive value = a / a + b X 100

11 / 30 x 100 = **36.67%**

Negative predictive value = c / c + d X 100

15 / 70 X 100 = **21.42%**

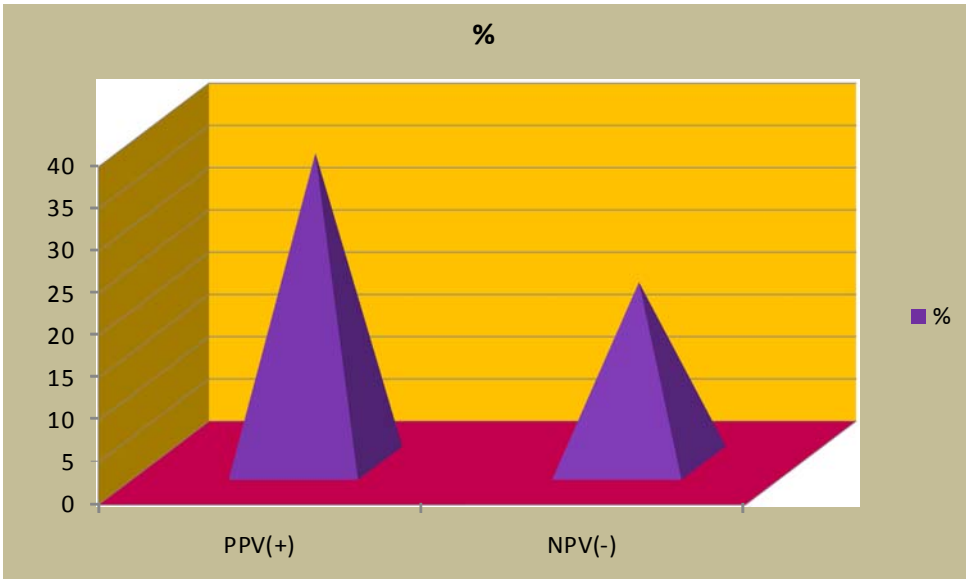
Sensitivity and specificity of DIPSI criteria



The sensitivity of DIPSI was 42.3%

The specificity of DIPSI was 74.32 %

Positive and Negative predictive value of DIPSI criteria



The Positive Predictive Value (PPV) of DIPSI is = 36.67 % .

The Negative Predictive Value (NPV) of DIPSI is = 21.42 % .

10. CORRELATION BETWEEN BLOOD GLUCOSE LEVEL (DIPSI CRITERIA) AND BMI

Table – 10

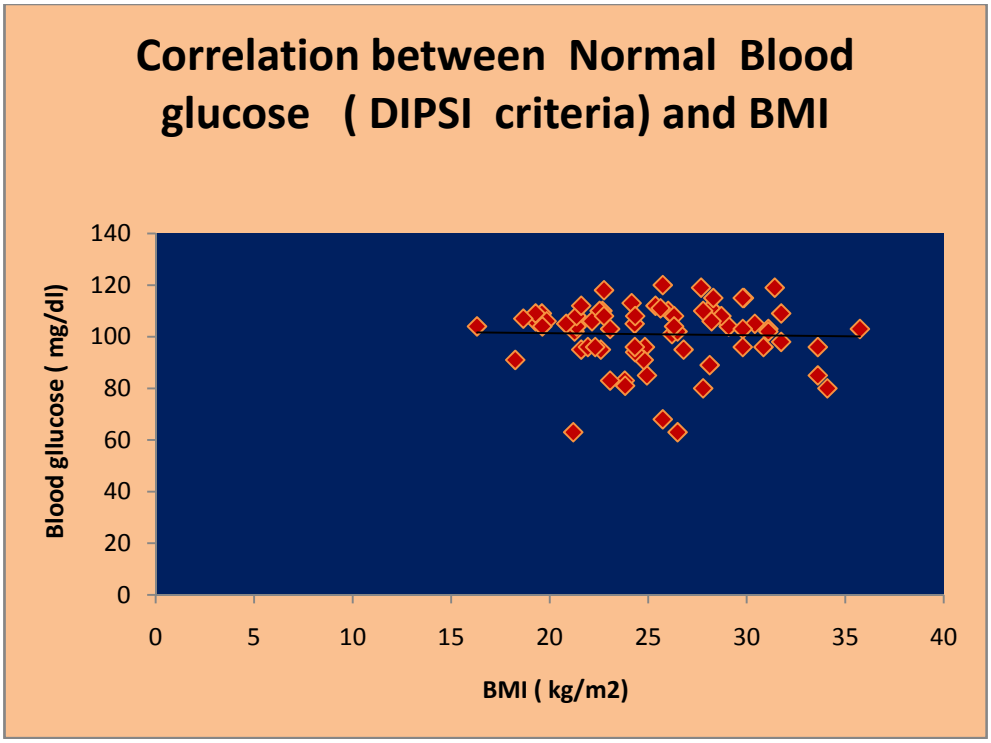
BMI	'r Value	p Value	95% C.I
Normal	-0.0279	0.4086	-0.259 To 0.206
Impaired Glucose Tolerance	-0.12933	0.2782	-0.514 To 0.298
Gestational Diabetes Mellitus	-0.25943	0.3098	-0.884 To 0.699

'r-=Correlation coefficient

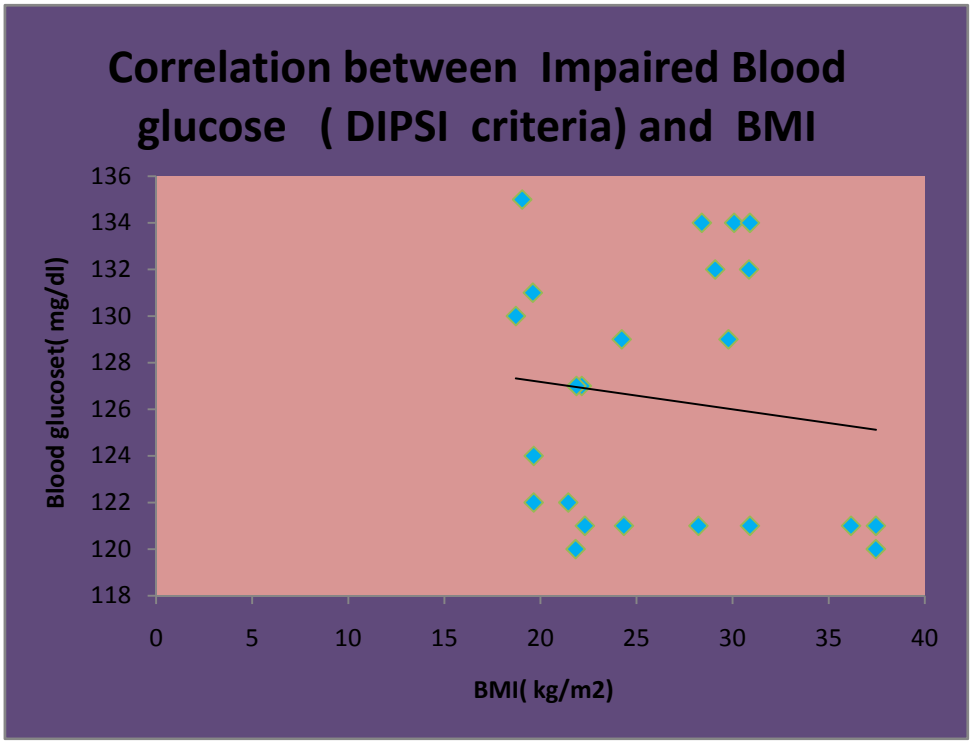
C.I-=confidence Interval

-r = Negative correlation

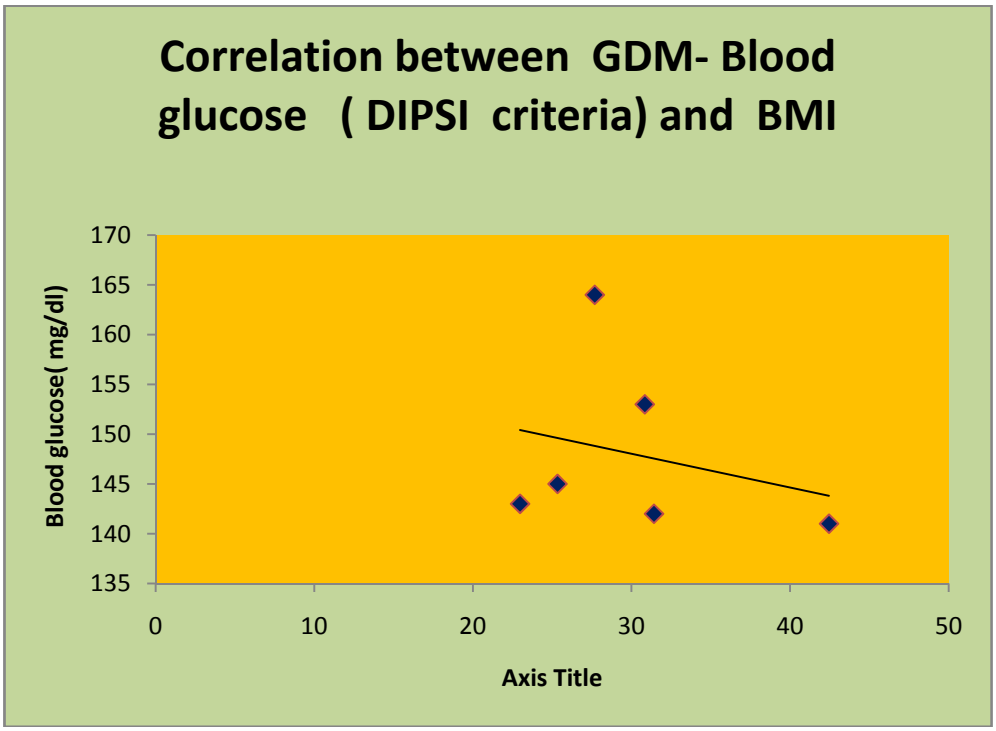
NS= Not significant



Of the 71 patients with normal blood glucose values there was a significant negative correlation (p value 0.4086) in comparison to BMI .



Of the total 23 patients with impaired glucose tolerance there was a significant negative correlation (p value = 0.2782) in comparison to their BMI.



Of the total 6 patients with Gestational Diabetes Mellitus there was a significant negative correlation (p value = 0.3098) in comparison to their BMI.

11. CORRELATION BETWEEN BLOOD GLUCOSE LEVEL (WHO CRITERIA) AND BMI

Table – 11

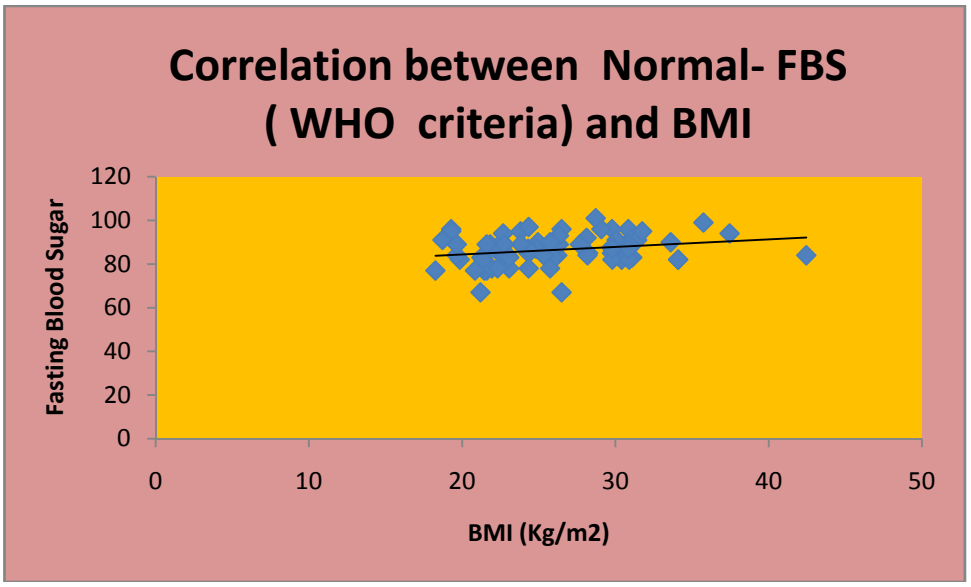
BMI	'r Value	p Value	95% C.I
Normal	Fasting=0.2518	0.014 *	0.027 to 0.452
	PPBS=0.2498	0.01532*	0.025 to 0.451
Impaired glucose tolerance	Fasting=0.5821	0.00898*	0.122 to 0.836
	PPBS = -0.0399	0.4416*	-0.525 to 0.464
Gestational Diabetes Mellitus	Fasting=0.03629	0.463*	-0.643 to 0.683
	PPBS=0.46106	0.1058	-0.861 to 0.962

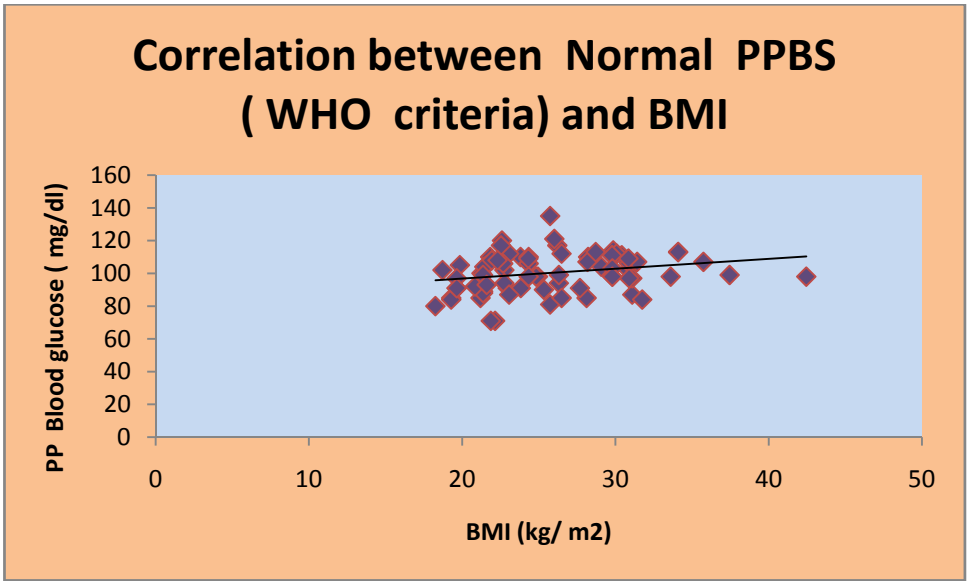
'r-=Correlation coefficient

C.I-=confidence Interval

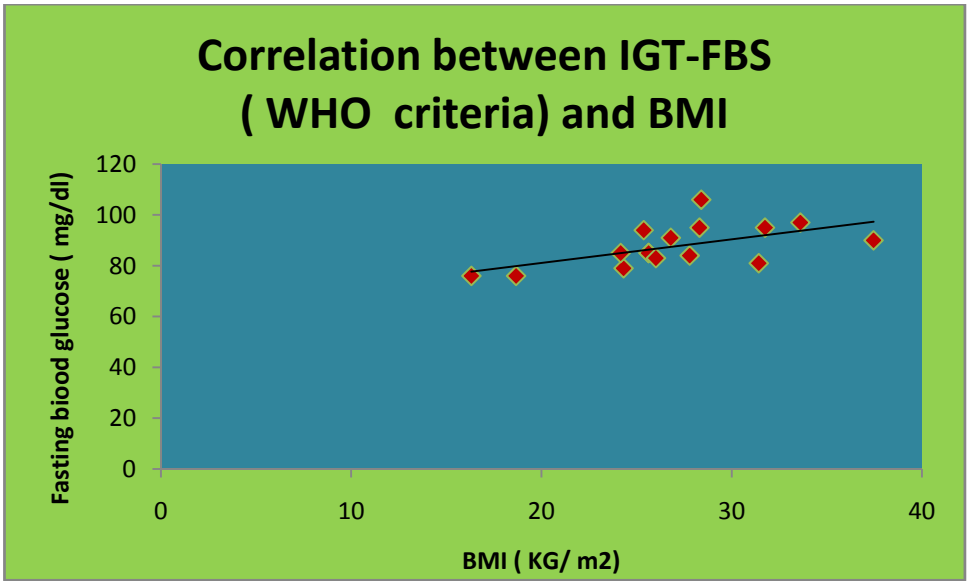
-r = Negative correlation

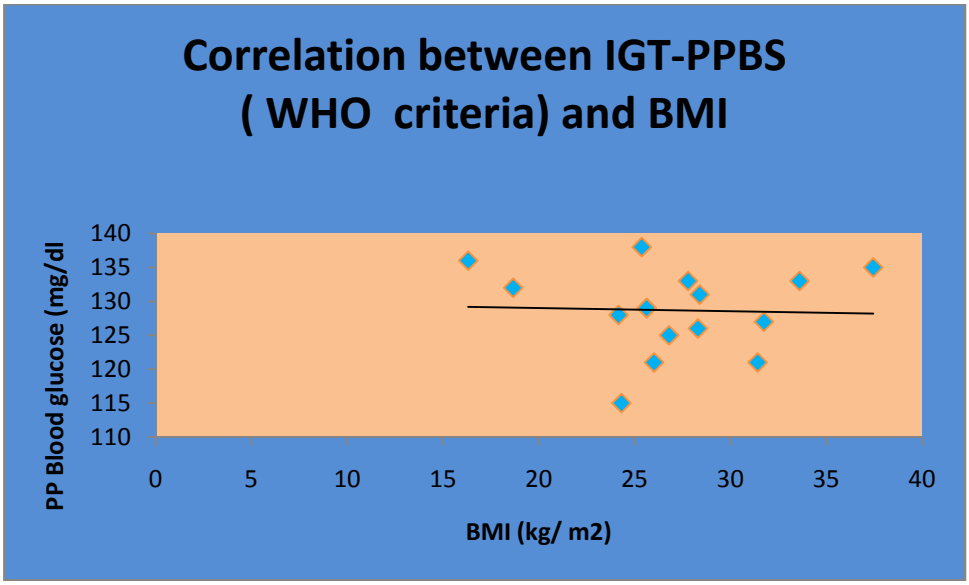
NS= Not significant



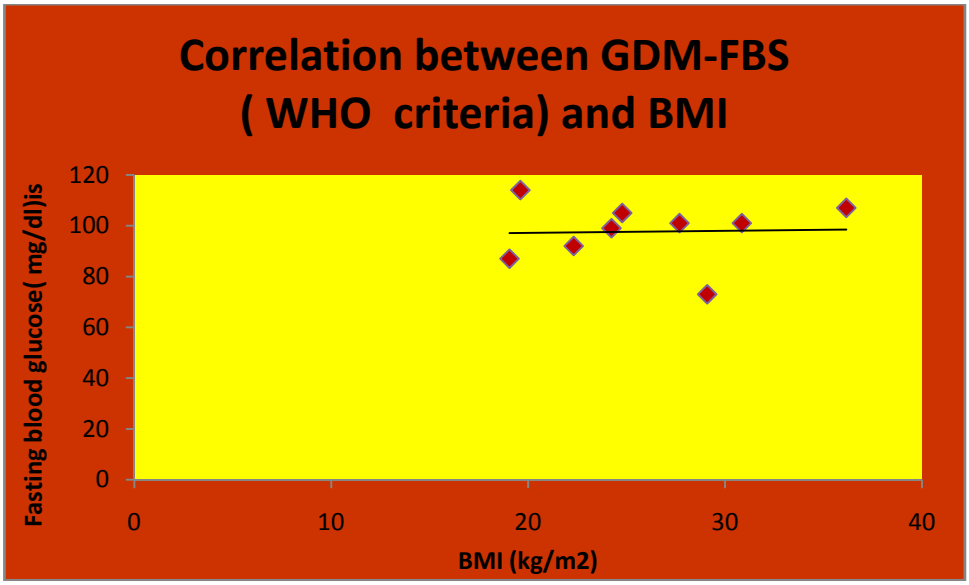


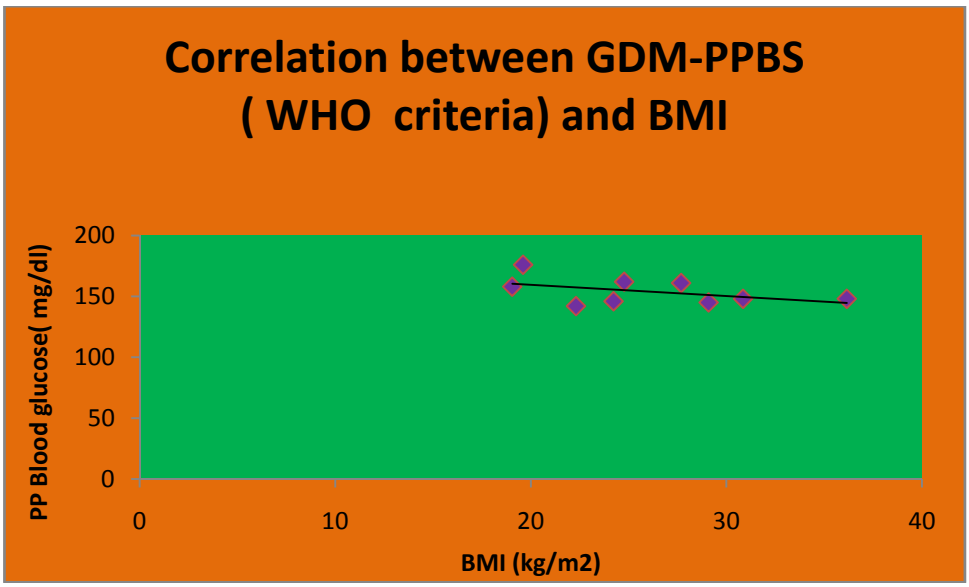
Of the total 75 patients with Normal FBS & PPBS in the WHO criteria in comparison to their BMI had a significant positive correlation. (p values = 0.014 & 0.01532 respectively).





Of the total 16 patients with impaired FBS & PPBS in the WHO criteria, in comparison to their BMI, FBS had a significant positive correlation (p values = 0.00898), Whereas,PPBS had a significant negative correlation (p values = 0.4416).





Of the total 9 patients with GDM, the FBS & PPBS in the WHO criteria in comparison to their BMI had a significant positive correlation. (p values = 0.463 & 0.1058respectively)

12. CORRELATION OF AGE AND BLOOD GLUCOSE (DIPSI CRITERIA)

Table – 12

Parameter	'r' value	'p' value	95% CI of 'r
Blood glucose	0.163095	0.0525*	-0.034 to 0.348

*- SIGNIFICANT

R =Correlation coefficient

C.I =confidence Interval

There is a significant positive correlation between DIPSI and Age (p value =0.0525) .

Discussion

Discussion

A prospective cross sectional study was conducted in PSG Hospital, Coimbatore in the Department of Obstetrics & Gynaecology during September 2013 – 2014.

A total number of 108 antenatal women between 24-28 weeks of gestation were enrolled in the study. Of the total 52 were primis and 48 were multi. All the were patients selected according to the exclusion and inclusion criteria. Overt Diabetes, type 1 DM, and patients on steroids were exclused from the study . Each patient underwent both WHO & DIPSI testing within a ten day interval period so that diet control would not falsely alter the values during the second testing. Advantages of DIPSI was that Fasting status was not required hence it does not alter her routine activities. It can serve as a both screening and diagnostic test, unlike the WHO recommendation which is a two step procedure and required the overnight fasting status. In pregnancy unlike other state fasting is very cumbersome procedure.

On the first visit FBS followed by 75 gms oral glucose test and 2hr later blood glucose testing according to the WHO criteria.

On the second visit 75gms glucose and 2hrs later blood glucose alone irrespective of the last meal according to the DIPSI criteria.

There were totally 8 defaulters in my study, out of which 4 had vomiting following glucose ingestion all the two times. And 3 were defaulters who did not undergo both testing hence excluded. One patient had betamethasone injection for fetal lung maturity at 28 weeks after the first test, hence had abnormally high value during the second testing so excluded from the study.

Our aim is to evaluate if a single blood test report after a 2hr oral glucose load irrespective of the last meal according to the DIPSI criteria can be used as a diagnostic test for Gestational diabetes mellitus and compare the values with that of the WHO recommendation which is a two step process which consists of a FBS followed by 2hrs later 75gms oral glucose test blood test.

The mean age of our patients were 26 yrs. There were 14 patients in the underage category (less than 20 yrs).64 patients were between 20-19yrs,17 patients between 30-34 yrs and 5 patients greater than 35 yrs.

The average FBS and 2hr PPBS according to WHO criteria were 87 and 109 mg/dl respectively and for DIPSI it was 109 mg/dl.

There were totally 52 primis and 48 multis. Out of the 100 patients recruited in this study there was 1 patient belonging to the underweight category(less than 40 kg), 41 to 50 kgs- 12 patients, 51 to 60 kgs- 42 patients, 61 to 70 kgs- 23 patients, 71to 80 kgs-17 patients,81 to 90kgs-1 patient,91 to 100- 4 patients. The average weight was 52 kgs.

The average BMI was 25 kg/m2. Out of which 14 patients belonged to the low BMI group of less than 20, 34 patients with BMI 21-24 kg/m2, 38 patients with BMI 25-30 kg/m2, 10 patients with BMI 31-35 kg/m2,and 4 patients BMI greater than 35 kg/m2. In the high BMI category >30kg/m2 there were 14 patients out of which 4 patients were positive in both DIPSI & WHO criteria. 2 patients had GDM according to WHO and 3 patients had GDM according to DIPSI criteria. 4 patients had normal blood sugar in both the criteria's.

Of the risk factors evaluated the majority of the patients who had Hypothyroid and previous LSCS has positive blood glucose values. Obesity had a risk of 10%, followed by previous

GDM 7%, elderly primi 5%, hypertensive disorders in pregnancy 4%. Others had 25% which included Anaemia 2%, Rh issoimunisation 1%, chronic hypertension 1%, Bad obstetric history 3%, asthma (not on steroids) 2% and 16% with no risk factors.

The correlation between blood glucose level in the WHO criteria and BMI was analysed by Spearman Rank correlation with Scatter diagram and found that there was a significant positive correlation (p value 0.014) between FBS and BMI & PPBS and BMI (p value 0.01532) in the normal category group.

In the Impaired glucose tolerance group there was a significant positive correlation(p value0.00898) between FBS and BMI. Only the PPBS and BMI in this study had a negative correlation (r value -0.0399). In the GDM group there was significant positive correlation between BMI & FBS/PPBS.

In the DIPSI group all the three values Normal/ impaired glucose tolerance/ GDM has only negative correlation with BMI.

Of the 100 patients enrolled in our study 75 patients had normal blood glucose according to the WHO and 71 had normal blood glucose according to DIPSI. The number of patients

categorized under impaired glucose tolerance according to WHO & DIPSI were 16 and 23 respectively. And finally the total number who were diagnosed as GDM under WHO & DIPSI were 9 and 6 respectively. Of the total only one patient with GDM was positive under both criteria's. 4 patients were positive for GDM only by DIPSI but had normal sugars by the WHO. 7 patients who tested positive for GDM under WHO were categorized as impaired glucose tolerance under DIPSI.

The true positives in our study were 11, and true negatives were 55. False positives were 19 and false negatives 15. Out of the 15 false negatives 2 patients had both FBS and PPBS values abnormal. There were totally 3 patients with abnormal Fasting values and 14 patients with abnormal PPBS values. Using this the sensitivity of DIPSI was calculated against WHO and was 42.3% . The specificity of DIPSI was 74.32%. The positive predictive value and negative predictive value was 36.67% and 21.42% respectively.

Further correlation and positivity of the study will have to evaluated after delivery and neonatal follow up .

Conclusion

CONCLUSION

Gestational diabetes is also found in patients without any risk factors, hence the need of universal screening.

BMI and age(elderly primi) had a significant positive correlation in our study.

Accelerated insulin catabolism by renal and placental insulinases and the anti-insulin effects of other hormones (cortisol, estriol, progesterone) produced in large amounts during pregnancy also contribute to insulin resistance. The increased insulin resistance in the third trimester explains why gestational diabetes is more common after 26 weeks.

Advantages of DIPSI was that Fasting status was not required hence it does not alter her routine activities. It can serve as a both screening and diagnostic test, unlike the WHO recommendation which is a two step procedure and required the overnight fasting status. In pregnancy unlike other state, fasting is very cumbersome procedure. And in a country like India where patients may not be available follow up for the 2 step or the 3 step procedure according to the IADPSG or

ADA guidelines DIPSI could serve as a diagnostic as well as a screening procedure.

The DIPSI (Diabetes in pregnancy study group India) recruited about 1463 pregnant women between April 2009- February 2010 and using the criteria following the 75gm glucose 2 hr test . Statistical analysis was done using chi-square test and independent-t test . The mean age was 23.60 ± 3.32yrs. The BMI was 21.5 ± 4.06 kg/m2 . The average age of gestation was 27.9 ± 5.56 weeks. This study showed 196 women which is 13.4% to have GDM and the remaining had normal values . They studied birth weight and followed up the neonate following treatment with either insulin or diet regulation and found out that macrosomia was seen in 9.9% GDM mothers and 9.8% in the normal blood glucose level mothers and there was no significant statistical difference between the GDM and normal blood glucose value mothers (p=1.000). However this DIPSI study did not have a control group which had untreated antenatal mothers with 2h PG $\geq$ 7.8 mmol/L (19).

International Association of The Diabetes and Pregnancy Study Group (IADPSG) and the WHO has many disadvantages such as the dropout rate is very high due to the necessity of fasting

blood sample .FPG values do not reflect the 2hr 75gm GCT which is supposedly the diagnostic test for GDM. Asian Indian ethnicity have high insulin resistance and so their 2hr PG is higher than Caucasians. Hence FPG is not an appropriate test for diagnosis of GDM(20). As a result FPG > 5.1mmol/L will miss 76% of GDM by the WHO criteria(21)

In a study which enrolled 1463 pregnant women who underwent IADPSG and DIPSI criteria. The objective of this study was to find out if DIPSI could diagnose GDM against the IADPSG. The prevalence of GDM with DIPSI was 13.4% (n=196) and IADPSG was 14.6% (n=214) and concluded that there was no statistical significance (P=0.21) between the two test and thereby implied a close agreement between the two tests.

In another Pilot study done GDM by Vijayalakshmi et al at PSIMS & RF in Andhra Pradesh which was to assess the effectiveness of DIPSI in diagnosing. This study enrolled 200 antenatal women who underwent DIPSI followed by the ADA criteria 3 days later out of the 200 women 22(11%) had abnormal DIPSI. And out of the 22 only 5 (2.5%) had abnormal ADA. As

a result 17 women were wrongly categorized had GDM by DIPSI. Thereby a prevalence of 2.5% was detected . The sensitivity being 100% and Specificity 89% and false positives 1.6% (22) .

IADPSG have included FBS and HbA1C in the first antenatal visit to all women or high risk women only according to the incidence of GDM, the rationale being fasting hyperglycemia supposedly is a more sensitive co-realtor of GDM which was justified by the HAPO guidelines .HAPO study demonstrated an increase in adverse perinatal outcome with elevation of FBS alone on OGTT. The physiological changes which occur during pregnancy the net effect is a lowering of maternal FBS with a sustained significant increase in post-prandial blood sugars. Hence for early and accurate diagnosis definitely FBS will have to be included in the criteria along with the 75gm glucose Post prandial .

Our study has a sensitivity of 42.3%, specificity of 74.32% and a positive predictive value of 36.67% .

Further follow up of the pregnancy until delivery to look for polyhydramnios mode of delivery, complication of DM in the mother,follow up of infant for LGA, Neonatal hypoglycemia and

other factors in neonate will be useful to substantiate the need for inclusion of FBS in the criteria.

Long term follow up of the mother, to find out if the high blood sugar in pregnancy was only GDM or an early reflection of type 2 DM is needed. The laboratory results are standard as it is auto-analysed.

Appendix

PROFORMA

1. Name

2. Age

3. IP/OP Number

4. LMP – EDD –

5. Obst Score (Parity)

6. Gestational age - at the time of test I

   - at the time of test II

7. Risk factors (Obst /Medical)

8. Weight (in kg)

9. Height (in cm)

10. Test results – Test I (WHO regimen)

    – Test II (DIPSI Criteria)

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

Recognized by The Strategic Initiative for Developing Capacity in Ethical Review (SIDCER)
POST BOX NO. 1674, PEELAMEDU, COIMBATORE 641 004, TAMIL NADU, INDIA
Phone : 91 422 - 2598822, 2570170, Fax : 91 422 - 2594400, Email : ihec@psgimsr.ac.in

September 9, 2013

To
Dr Shruthii Nanjundappan
Postgraduate
Department of Obstetrics & Gynaecology
PSG IMS & R
Coimbatore

The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on 21st June, 2013 in its expedited review meeting held at College Council Room, PSG IMS&R, between 2.00 pm and 3.30 pm, and discussed your study proposal entitled:

"One step approach for diagnosis of gestational diabetes mellitus in pregnancy"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Informed Consent Form (Tamil)
4. Informed Consent Form (English Ver. 1.1)
5. Data Collection Tool
6. Budget
7. CV

After due consideration, the Committee has decided to approve the above study.

The members who attended the meeting, at which your proposal was discussed, are listed below:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member - Secretary	Female	Yes	Yes
Dr Sudha Ramalingam	M.D	Epidemiologist Alt. Member - Secretary	Female	Yes	Yes
Dr D Vijaya	Ph D	Member – Basic Scientist	Female	Yes	Yes
Dr Y S Sivan	Ph D	Member – Social Scientist	Male	Yes	Yes

The approval is valid for one year.

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

Recognized by The Strategic Initiative for Developing Capacity in Ethical Review (SIDCER)
POST BOX NO. 1674, PEELAMEDU, COIMBATORE 641 004, TAMIL NADU, INDIA
Phone : 91 422 - 2598822, 2570170, Fax : 91 422 - 2594400, Email : ihec@psgimsr.ac.in

We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

This Ethics Committee is organized and operates according to Good Clinical Practice and Schedule Y requirements.

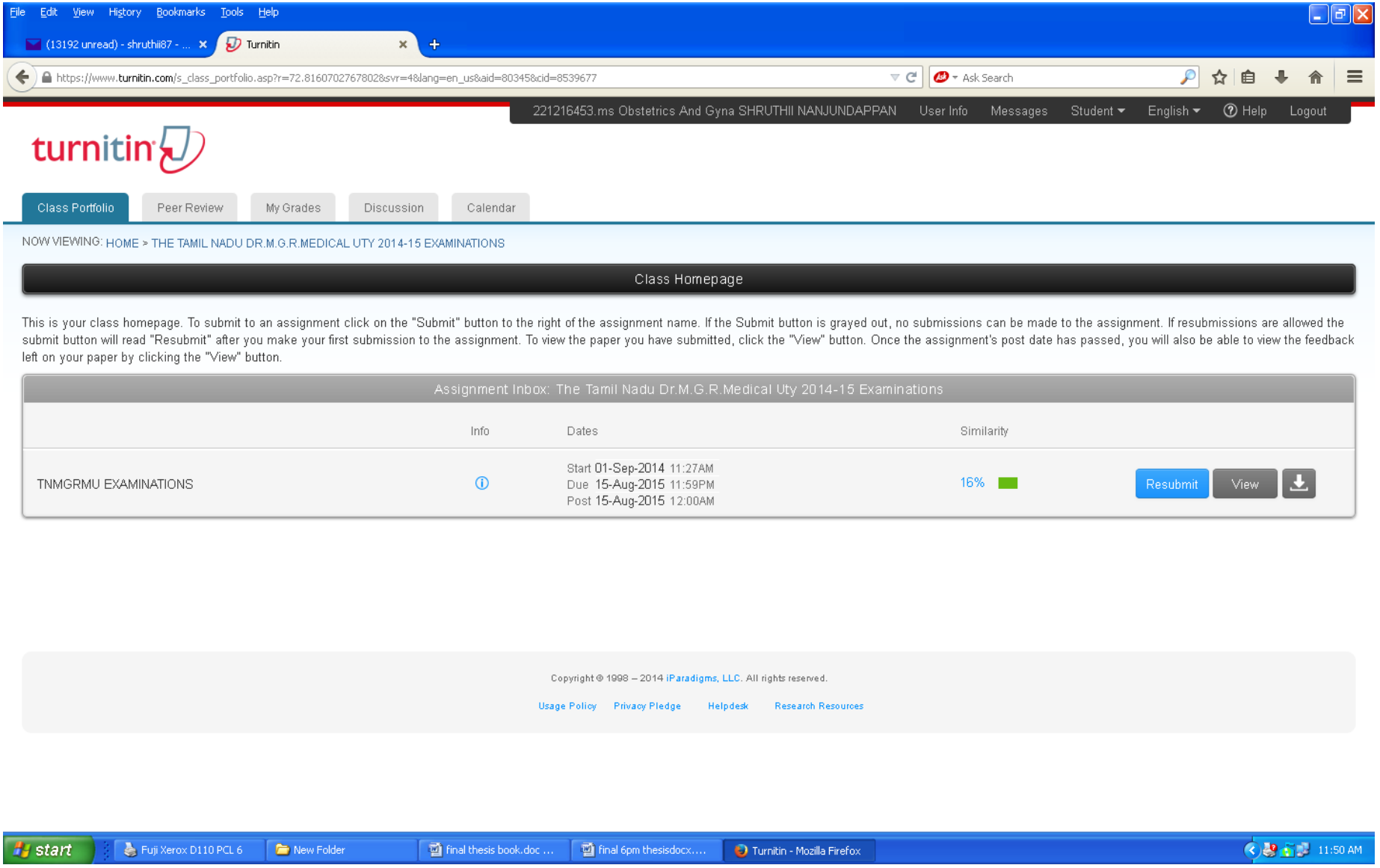
Non-adherence to the Standard Operating Procedures (SOP) of the Institutional Human Ethics Committee (IHEC) and national and international ethical guidelines shall result in withdrawal of approval (suspension or termination of the study). SOP will be revised from time to time and revisions are applicable prospectively to ongoing studies approved prior to such revisions.

Yours truly,

10.9.13

Dr S Bhuvaneshwari
Member - Secretary
Institutional Human Ethics Committee

SECRETARY
PSG IMS & R
COIMBATORE-641 004
INSTITUTIONAL HUMAN ETHICS COMMITTEE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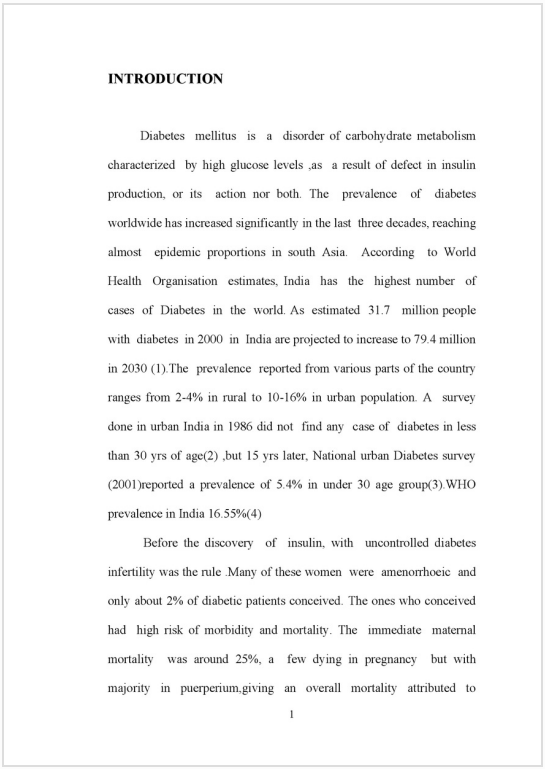

INTRODUCTION

Diabetes mellitus is a disorder of carbohydrate metabolism characterized by high glucose levels ,as a result of defect in insulin production, or its action nor both. The prevalence of diabetes worldwide has increased significantly in the last three decades, reaching almost epidemic proportions in south Asia. According to World Health Organisation estimates, India has the highest number of cases of Diabetes in the world. As estimated 31.7 million people with diabetes in 2000 in India are projected to increase to 79.4 million in 2030 (1).The prevalence reported from various parts of the country ranges from 2-4% in rural to 10-16% in urban population. A survey done in urban India in 1986 did not find any case of diabetes in less than 30 yrs of age(2) ,but 15 yrs later, National urban Diabetes survey (2001)reported a prevalence of 5.4% in under 30 age group(3).WHO prevalence in India 16.55%(4)

Before the discovery of insulin, with uncontrolled diabetes infertility was the rule .Many of these women were amenorrhoeic and only about 2% of diabetic patients conceived. The ones who conceived had high risk of morbidity and mortality. The immediate maternal mortality was around 25%, a few dying in pregnancy but with majority in puerperium,giving an overall mortality attributed to

1

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PSG Institute of Medical Science and Research, Coimbatore
Institutional Human Ethics Committee
INFORMED CONSENT

I , Dr.Shruthii Nanjundappan MS.,(OG) postgraduate from the department of Obstetrics and Gynecology of PSG Institue of Medical Science & Research (PSGIMS&R), am carrying out a study on the topic: " One step approach for diagnosis of gestational diabetes mellitus" to under the aegis of the Department of Obstetrics and Gynecology, PSGIMSR

The objectives of this study are: To estimate the sensitivity and specificity of the DIPSI criteria

Sample size: 100 Respondants are term antenatal patients between 23-29weeks who come to the OP Departmant of Obstetrics & Gynacology in PSG hospitals – , Coimbatore

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator/s. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study.

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date: Witness:

Master Chart

SL.No	Name	Age	OP Number	LMP/EDD	Obstetric Score	GA I	GA II	Risk Factors	Weight	Height	Height/ 100	(height /100)^2	BMI	Results I	Results II
1	Najimunisha	33	O14003345	28-8-13/4/6/14	G2P1L1	26W	26W+4D	Prev. LSCS	72.8	153	1.53	2.3409	31.0991499	83 / 87	103
2	Bhuvaneshwari	21	O13090395	15-10-13/22-7-14	G2P1L0A0	25W+2D	26W	NND,no live child	47.7	150	1.5	2.25	21.2	67 / 85	63
3	Priya	25	O13050235	4-12-13/11-9-14	G2A1	22W	24W		59.4	153	1.53	2.3409	25.37485582	94 / 138	112
4	Manimegala	23	O12013118	10-11-13/17-8-14	G2P1L1	25W+6D	26W+6D	GDM on diet	60.3	162	1.62	2.6244	22.97668038	84 / 91	143
5	Suguna . S	34	O14030428	17-10-13/4-9-14	G4P1L1A2	24W	26W	GDM on OHA	70	159	1.59	2.5281	27.68877813	101 / 161	164
6	Ambikadevi	27	O14008151	4-12-13/11-9-14	G2P1L1	26W+6D	27W+5D	Prev LSCS	57.3	162	1.62	2.6244	21.83356196	89 / 110	120
7	Kanimozhi	29	O07065729	18-12-13/25-9-24	G3P1L1A1	27W+1D	24W		72.2	153	1.53	2.3409	30.84283822	96 / 102	153
8	Vidhya	25	O14010604	14-11-13/21-8-14	Primi	25W	26W		69.9	153	1.53	2.3409	29.86031014	86 / 114	115
9	Sameera Banu	33	O14016772	1-10-13/8-7-14	G4P2L2A1	23W+2D	24W		70.3	137	1.37	1.8769	37.45537855	90 / 135	120
10	Rukkumani	20	O14020461	21-10-13/28-7-14	Primi	24W+2D	26W		58.4	153	1.53	2.3409	24.9476697	90 / 98	85
11	Nithya	22	O14026538	28-1-14/5-11-14	Primi	24W	24W+1D		72.7	160	1.6	2.56	28.3984375	106 / 131	134
12	Sangeetha	26	O13083050	23-3-14/30-12-14	Primi	23W+4D	24W		62	158	1.58	2.4964	24.8357635	87 / 98	96
13	MohanaPriya	26	O13064564	6-11-13/13-8-14	Primi	25W	26W		65.2	152	1.52	2.3104	28.22022161	85 / 110	121
14	Lavanya	23	O14012409	18-10-13/25-7-14	Primi	26W+4D	27W+5D		53.8	149	1.49	2.2201	24.23314265	99 / 146	129
15	Tamil Arasi	24	O14009938	20-11-13/27-8-14	Primi	29W	30W		58	153	1.53	2.3409	24.77679525	105 / 162	91
16	Rubinya	27	O14009640	7-11-13/14-8-14	G3P1L1A1	26W+2D	27W+5D		54.3	151	1.51	2.2801	23.81474497	95 / 110	83
17	Rani	32	O14007941	8-12-13/15/9/14	G2P1L1	24W	25W	Rh-ve,prev LSCS	50	161	1.61	2.5921	19.28937927	96 / 85	106
18	Nazexin Sulthan	34	O14003380	20-9-13/27-6-14	G2P2L2	25W+3D	27W		68.5	157	1.57	2.4649	27.79017404	84 / 133	80
19	Preethi	23	O09052316	24-11-3/31-8-14	Primi	23W	24W	SVT	61.6	148	1.48	2.1904	28.12271731	92/ 85	89
20	Radhika	26	O14002950	16-11-13/3-9-14	Primi	24W+1D	25W	subclinical hypothyroid	60.9	147	1.47	2.1609	28.18270165	84 / 107	111
21	Jothi	23	O14019604	17-12-13/26-9-14	G2P1L0A0	24W	25W+1D	NND,no live child	67.1	160	1.6	2.56	26.2109375	84 / 117	101
22	Maheshwari	20	O14012731	14-8-13/21-5-14	G2A1	27W	28W		56	159	1.59	2.5281	22.15102251	87 / 71	127
23	Jayapriya	25	O14011201	25-7-13/2-5-14	Primi	27W	28W		60.9	153	1.53	2.3409	26.01563501	83 / 121	110
24	Soorya	21	O14033805	7-1-14/14-10-14	Primi	24W+3D	24W+5D		45	157	1.57	2.4649	18.25631871	77/80	91
25	Jenafar	25	O12035366	26-7-13/3-5-14	Primi	28W	29W+6D		50	162	1.62	2.6244	19.05197378	87/158	135
26	Nageshwari	18	O13074469	13-8-13/20-5-14	Primi	27W+1D	27W+2D		57.2	164	1.64	2.6896	21.26710291	83/100	102
27	Saranya	26	O13075875	27-8-13/3-6-14	G2P1L1	26W+6D	28W	Prev LSCS	67.9	147	1.47	2.1609	31.42209265	81/121	142
28	Anandhi	27	O09039761	24-8-13/31-5-14	G2P1L1	26W+1D	27W		65.4	164	1.64	2.6896	24.3158834	79/115	105
29	Radha	28	O09053085	30-7-13/6-5-14	G2P1L1	24W+1D	26W	Prev LSCS	46.5	154	1.54	2.3716	19.60701636	114/176	109
30	Sankara lakshmi	27	O09030287	15-12-13/22-13-14	G3P2L2	23W+2D	27W	Prev LSCS, Anemia	64.2	163	1.63	2.6569	24.16349881	85/128	113
31	Blessy	30	O13093273	10-7-13-17-4-14	Primi	24W	25W	ITP	53.8	133	1.33	1.7689	30.41438182	82/111	105
32	Selvi	29	O06025762	4-11-13/11-8-14	G3P1L1	28W	29W	Prev LSCS, Prev Ectopic	78.3	148	1.48	2.1904	35.74689554	99/107	103
33	Jeevitha	26	O14002257	28-8-13/4-6-14	Primi	25W	26W		55.4	161	1.61	2.5921	21.37263223	79/89	106
34	Shanthi	35	O14004337	22-8-13/29-5-14	Primi	24W	25W	Elderly primi, Hypothyroid	56	161	1.61	2.5921	21.60410478	77/105	95

35	Velumani	24	O14029873	29-11-13/6-9-14	Primi	23W+6D	25W		52.2	156	1.56	2.4336	21.44970414	77/104	122
36	Suganthi	24	O14001926	2-9-13/9-6-14	Primi	27W	28W		53.81	136	1.36	1.8496	29.09277682	73/145	132
37	saima	26	O13009332	28-8-13/4-6-14	Primi	25W	26W	Hypothyroid	55.4	161	1.61	2.5921	21.37263223	79/88	104
38	Mutulakshmi	20	O14011257	21-8-13/28-5-14	Primi	24W+1D	26W		60	153	1.53	2.3409	25.6311675	85/129	111
39	Shobana	28	O12018053	23-7-13/30-4-14	G2A1	24W+1D	24W+2D		60	151	1.51	2.2801	26.31463532	89/94	108
40	maheshwari	27	O14012731	8-12-13/13-9-14	G2P1L1	24W	25W		50	161	1.61	2.5921	19.28937927	95/84	109
41	Maheshwari	20	O14012731	14-8-13/21-5-14	G2A1	27W	28W		55.3	159	1.59	2.5281	21.87413473	87/71	127
42	Yamuna	20	O14011721	13-10-13/20-7-14	Primi	26W	28W	Hypothyroid	55	156	1.56	2.4336	22.60026298	90/120	95
43	Mohana Priya	27	O13064564	6-11-13/13-8-14	Primi	25W	26W		60	157	1.57	2.4649	24.34175829	85/110	121
44	Srividhya	21	O13090111	13-10-13/20-7-14	Primi	26W	27W		52.1	162	1.62	2.6244	19.85215668	82/105	106
45	Lavanya	28	O14009873	3-1-13/1-10-14	Primi	25W+5D	26W	Hypothyroid, Anemia	80.1	164	1.64	2.6896	29.78138013	85/111	129
46	jayanthi	37	O13072388	28-8-13/4-6-14	G3A2	25W+2D	26W	BOH, Elderly primi, APLA +ve	88	156	1.56	2.4336	36.16042078	107/148	121
47	saranya	39	O13079355	4-12-13/11-9-14	G2P1L1	26W+6D	27W+5D	long period of infertility	78	159	1.59	2.5281	30.85320992	101/148	132
48	Mahadevi	23	O13069402	12-1-14/19-10-14	Primi	24W+4D	26W		37.2	151	1.51	2.2801	16.3150739	76/136	104
49	Kayalvizhi	24	O14019449	21-1-14/28-10-14	Primi	23W+2D	25W		55	157	1.57	2.4649	22.31327843	92/142	121
50	Kalpana	22	O14018627	16-4-14/23-10-15	Primi	23W+4D	25W		60	157	1.57	2.4649	24.34175829	78/106	94
51	Revathi	30	O14014863	15-1-14/22-10-14	Primi	24W+5D	25W+1D	long period of infertility	67.2	152	1.52	2.3104	29.08587258	96/104	104
52	Nasreen Fathima	22	O14028771	Not knoown/19-12-14	G2A1	24W	26W		66.9	158	1.58	2.4964	26.79858997	91/125	95
53	Priya	29	O10098081	3-1-14/6-11-14	G4P1L1A2	25W+6D	26W	Prev LSCS	59	161	1.61	2.5921	22.76146754	83/102	118
54	Gnanpriya	21	O13057303	22-1-14/29-10-14	Primi	25W	26W		72.6	159	1.59	2.5281	28.71721846	101/113	108
55	Shabara	26	O11062679	26-11-14/4-11-14	G2P2L1	23W	24W	Prev LSCS	75.3	154	1.54	2.3716	31.75071682	95/127	98
56	Vyshnavi	21	O14047418	10-1-14/17-10-14	Primi	26W+6D	27W+6D		44.4	154	1.54	2.3716	18.7215382	91/102	130
57	Padmavathy	22	O13020079	30-1-14/6-11-14	Primi	25W	24W		59.2	156	1.56	2.4336	24.32610125	87/109	96
58	Saranya	19	O12053436	10-2-14/17-11-14	G2P1L1	24W	24W+4D	Prev LSCS	53.4	156	1.56	2.4336	21.94280079	78/108	96
59	Nithya	22	O14026538	28-1-14/4-11-14	Primi	26W	27W		77	160	1.6	2.56	30.078125	91/112	134
60	Ramya	28	O09070652	6-1-14/13-10-14	G2P1L1	28W	28W+3D	Prev LSCS	65.4	152	1.52	2.3104	28.3067867	95/126	115
61	Jenitamary	20	O12010047	18-6-13/25-3-14	G2P1L1	28W+6D	29W	bronchial asthma, Obesity	93	148	1.48	2.1904	42.45799854	84/98	141
62	Sajitha	19	O13074493	24-1-14/31-10-14	G3P1L1A1	25W	26W	bronchial asthma, Prev LSCS	53.3	152	1.52	2.3104	23.06959834	78/112	83
63	Banupriya	24	O14019841	30-1-14/6-10-14	Primi	25W+1D	25W+4D		51.5	147	1.47	2.1609	23.83266232	89/91	81
64	Sangeetha	23	O14030978	22-12-13/29-9-14	Primi	29W+1D	30W		91.7	164	1.64	2.6896	34.09428911	82/113	80
65	Priya	23	O14013261	8-1-14/13-10-14	G3P1A1	24W+4D	28W	BOH, Prev IUD	46	153	1.53	2.3409	19.65056175	89/91	122
66	Rajeshwari	28	O14010743	25-8-13/1-6-14	G2P1L1	26W	27W	prev LSCS	52.4	152	1.52	2.3104	22.6800554	94/106	110
67	Parvathy Priya	21	O14018435	26-1-14/2-11-14	Primi	25W+1D	26W		57.7	160	1.6	2.56	22.5390625	87/117	110
68	Alphonsa	24	O14048332	7-12-13/14-9-14	Primi	28W+5D	29W		54.7	162	1.62	2.6244	20.84285932	77/92	105
69	Karthika	26	O13055628	9-1-14/16-10-14	G2A1	29W	29W+1D		65.3	157	1.57	2.4649	26.49194693	96/112	102

	Senthilkumar														
70	Padmapriya	26	O14034205	10-2-14/17-11-14	G2A1	24W	24W+1D	DCDA Twins	71.6	155	1.55	2.4025	29.80228928	88/103	115
71	Rajeshwari	28	O14028534	19-1-14/26-10-14	Primi	27W+1D	28W	Rh -ve	66.5	162	1.62	2.6244	25.33912513	84/90	145
72	Suganya	24	O14023592	22-2-14/29-11-14	Primi	23W	24W		65.9	160	1.6	2.56	25.7421875	78/81	68
73	Poonkodi	32	O11065327	14-10-13/27-7-14	G2P1L1	26W	27W	Chr.Hypertension	75.2	156	1.56	2.4336	30.90072321	89/97	134
74	Sangeetha	32	O14028316	3-2-14/10-11-14	G3P2L1A1	25W	26W	Fibroid	54.7	160	1.6	2.56	21.3671875	82/99	108
75	Gayathri	24	O14032198	12-3-14/19-12-14	G2P1L1	23W	24W	Prev LSCS	91.5	165	1.65	2.7225	33.60881543	97/133	96
76	Ananthi	24	O05013590	15-4-14/22-1-15	G2P1L1	23W	24W	hypothyroid	43	148	1.48	2.1904	19.6311176	91/107	104
77	samundeshwara	22	O13008764	14-10-13/21-7-14	Primi	26W	27W	Hypothyroid	75.2	156	1.56	2.4336	30.90072321	82/106	121
78	Radha	32	O1174338	19-1-14/26-10-14	Primi	27W+1D	28W	hypothyroid	71.6	155	1.55	2.4025	29.80228928	82/98	103
79	Gokilaveni	36	O08070215	8-1-14/15-10-14	G2P1L1A1	24W+5D	29W		46	153	1.53	2.3409	19.65056175	84/97	124
80	Sandya	17	O13091254	12-1-14/19-10-14	Primi	24W+4D	26W		42	150	1.5	2.25	18.66666667	76/132	107
81	yamuna	33	O14001174	28-8-13/4/6/14	G2P1L1	26W	26W+4D	Prev. LSCS	72.8	153	1.53	2.3409	31.0991499	92/97	102
82	mohanavidya	34	O10041401	17-10-13/4-9-14	G4P1L1A2	24W	26W	GDM on OHA	70	159	1.59	2.5281	27.68877813	89/91	119
83	fathima	33	O13093084	1-10-13/8-7-14	G4P2L2A1	23W+2D	24W		70.3	137	1.37	1.8769	37.45537855	94/99	121
84	logeshwri	26	O01088737	6-11-13/13-8-14	Primi	25W	26W		65.2	152	1.52	2.3104	28.22022161	88/89	106
85	sheeba	34	O14011876	20-9-13/27-6-14	G2P2L2	25W+3D	27W		68.5	157	1.57	2.4649	27.79017404	78/92	110
86	deepa	20	O14005319	14-8-13/21-5-14	G2A1	27W	28W		56	159	1.59	2.5281	22.15102251	79/87	106
87	subashini	26	O14011769	27-8-13/3-6-14	G2P1L1	26W+6D	28W	Prev LSCS	67.9	147	1.47	2.1609	31.42209265	97/109	119
88	bagyaveni	28	O14011927	30-7-13/6-5-14	G2P1L1	24W+1D	26W	Prev LSCS	46.5	154	1.54	2.3716	19.60701636	94/99	131
89	poongodi	35	O13040392	22-8-13/29-5-14	Primi	24W	25W	Elderly primi, Hypothyroid	56	161	1.61	2.5921	21.60410478	89/93	112
90	thangamani	28	O13007804	23-7-13/30-4-14	G2A1	24W+1D	24W+2D		60	151	1.51	2.2801	26.31463532	93/99	104
91	sangeetha	27	O13070900	6-11-13/13-8-14	Primi	25W	26W		60	157	1.57	2.4649	24.34175829	97/98	108
92	gulzar	39	O13085800	4-12-13/11-9-14	G2P1L1	26W+6D	27W+5D	long period of infertility	78	159	1.59	2.5281	30.85320992	87/109	96
93	vijayalakshmi	24	O13072688	21-1-14/28-10-14	Primi	23W+2D	25W		55	157	1.57	2.4649	22.31327843	78/108	96
94	indumathi	29	O13073354	3-1-14/6-11-14	G4P1L1A2	25W+6D	26W	Prev LSCS	59	161	1.61	2.5921	22.76146754	89/94	108
95	bindhu	26	O14009878	26-11-14/4-11-14	G2P2L1	23W	24W	Prev LSCS	75.3	154	1.54	2.3716	31.75071682	95/84	109
96	sureka	19	O13072485	24-1-14/31-10-14	G3P1L1A1	25W	26W	bronchial asthma, Prev LSCS	53.3	152	1.52	2.3104	23.06959834	83 / 87	103
97	ponsathyabama	26	O13084909	9-1-14/16-10-14	G2A1	29W	29W+1D		65.3	157	1.57	2.4649	26.49194693	67 / 85	63
98	Karthika	24	O08049511	22-2-14/29-11-14	Primi	23W	24W		65.9	160	1.6	2.56	25.7421875	90 / 135	120
99	kiruthika	24	O07006651	12-3-14/19-12-14	G2P1L1	23W	24W	Prev LSCS	91.5	165	1.65	2.7225	33.60881543	90 / 98	85
100	subaida	32	O13017546	19-1-14/26-10-14	Primi	27W+1D	28W	hypothyroid	71.6	155	1.55	2.4025	29.80228928	87 / 98	96

LIST OF ABBREVATIONS

GDM	-	Gestational Diabetes Mellitus
GCT	-	Glucose challenge test
OGTT	-	oral glucose tolerance test
FFA	-	free fatty acids
TG	-	triglycerides
AA	-	Amino acids
NST	-	Non stress test
BPP	-	Biophysical profile
ADA	-	American diabetic association
WHO	-	World Health Origination
ACOG	-	American College of Obstetricians & Gynecologist
NDDG	-	National Diabetes Data Group
TNF	-	Tumor Necrosis Factor
IDM	-	Infant of Diabetic Mother
PIH	-	Pregnancy induced hypertension
USG	-	Ultrasonography
FBS	-	Fasting Blood Sugar
PPBS	-	Post Prandial Blood Sugar
BMI	-	Body Mass Index
FPG	-	Fasting Plasma Glucose
PPG	-	Post Plasma Glucose
ANC	-	Antenatal care